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American Journal of Diseases of Children

VOL. 9 JUNE, 1915 No. 6

THE USE OF BISMUTH PILLS IN THE FLUOROSCOPIC EXAMINATION OF THE INFANT'S STOMACH *

ALFRED F. HESS, M.D.
NEW YORK

The use of pills for gastric tests suggested itself in the course of the employment of the duodenal catheter for ascertaining the size of the pylorus in infants. The size of the catheter which can be readily introduced past the pylorus in the newborn as well as in somewhat older infants[1]—the approximate circumferences of the pylorus at these periods of life—having been ascertained, it seemed that the same information could be obtained more easily through the use of pills having definite circumferences. Accordingly, bismuth pills were prepared of three sizes (9, 15 and 21 mm. in circumference), and coated with many layers of keratin to prevent their dissolution by the gastric juice.

Fig. 1.—Two-months-old baby. After one hour both pills are within stomach.

The use of pills has certain advantages over that of a powder suspended in milk, which is the usual way of giving bismuth to infants

* Received for publication March 12, 1914.

1. Hess, A. F.: The Pylorus, Pylorospasm and Allied Spasms, AM. JOUR. DIS. CHILD., 1914, vii, 184.

for radioscopic examination of the stomach. The introduction of the pill is very simple; it is placed on the dorsum of the tongue and pushed down gently into the pharynx by means of the finger. This would seem less harmful than pouring a considerable amount of bismuth mixture into the stomach. It was anticipated that the fluoroscopic picture would be more clearly defined with the use of the pill, and that we would be able to judge with greater certainty whether or not

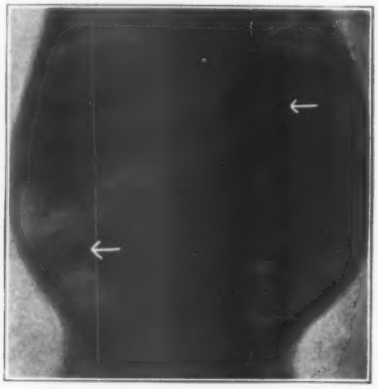

Fig. 2.—Same case as Figure 1. Large pill out of stomach; small pill in stomach after two hours.

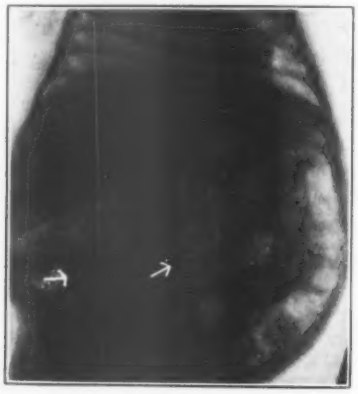

Fig. 3.—Same case as Figures 1 and 2. After three hours both pills out of stomach.

all the bismuth had left the stomach. Experience served to verify this opinion. When a suspension of bismuth is used, judgment is hampered frequently by a portion of the bismuth remaining in the stomach, while another portion has passed into the intestine.

Our first experiences soon convinced us, however, that we could not make use of these pills for the purpose of gauging the diameter of

the normal pylorus, for it was evident that the pills did not leave the stomach and enter the intestine according to their size. It was found that although a small and a large pill were given, for example, 9 F. or 15 F., there might be no difference in the time of their entering the intestine, as determined by fluoroscopic examinations carried out at half hour intervals. Moreover, if one did traverse the pylorus in advance of the other, it was almost always the larger and not the

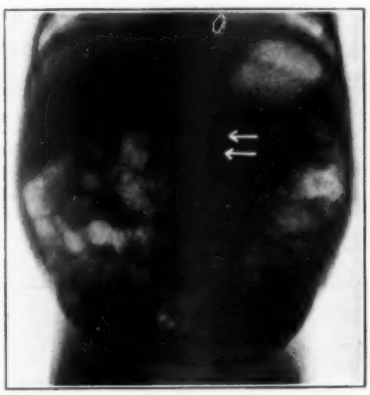

Fig. 4.—Six-months-old baby. After one hour both pills are within the stomach.

Fig. 5.—Same case as Figure 4. After two hours both pills are within the stomach, near pylorus.

smaller pill. Figures 1, 2 and 3 illustrate this observation. In this case, two pills—No. 9 and No. 15—were given. Roentgenogram 1 was taken one hour later, and shows both pills still within the stomach; Figure 2, taken after two hours, shows the larger pill out of the stomach and the smaller one still within the gastric cavity; Figure 3, taken after a three hour period, portrays both pills out of the stomach.

TABLE 1.—Showing Time of Passage of Pills from Stomach in Case 1
Ida C., aged 5½ months; dyspepsia, atrophy; weight, 11¼ pounds.

Date	Size of Pills	Given at	Fluoroscoped	Site of Pills	Summary	Remarks
10/16/14	9, 15	1:10 p. m.	2½ hours	Both in stomach	Out 2½-4 hrs.	
			4 hours	Both out of stomach		
10/20/14	9, 15	1:10 p. m.	2½ hours	Both out of stomach	Out within 2½ hrs.	
10/24/14	9, 21	1:15 p. m.	2 hours	No. 21 out and No. 9 in stomach	No. 21 out in 2 hrs.	Larger pill out earlier.
			3 hours	No. 21 out and No. 9 in stomach		
			3¾ hours	No. 21 out and No. 9 in stomach		
			4 hours	Both out of stomach	No. 9 out in 3¼-4 hrs.	
11/17/14	9, 15	10:00 a. m.	1 hour	Both in stomach	No. 15 out in 1 hr.	Larger pill out earlier.
			2 hours	No. 15 out and No. 9 in stomach	No. 9 out in 2-3 hrs.	
			3 hours	Both out of stomach		
11/21/14	9, 15	10:00 a. m.	1½ hours	Both in stomach		
			2½ hours	Both in stomach		
			3½ hours	Both out of stomach	Out 2½-3½ hrs.	

Epicrisis: Larger pill left stomach either at same time or earlier than smaller pill.

This result at first was quite surprising; further tests convinced us that it should be regarded as not exceptional, and that in cases manifesting no obstruction of the pylorus, where one pill does leave the stomach before the other, it will almost invariably be the larger one; for example, see Table 1, Case 1.

Frequently pills are noted to have left the stomach at about the same time, irrespective of their size. Figures 4, 5 and 6 show this result in roentgenograms taken one, two, and three hours following the introduction of a No. 9 and a No. 15 pill. The first and second show both pills within the stomach, and the third shows both in the intestine.[2] It is realized that the second roentgenogram is not convincing in this regard. But it should be borne in mind that this study

Fig. 6.—Same case as Figures 4 and 5. After three hours both pills out of stomach; one disintegrating.

was carried out, not by means of roentgenography, but almost entirely with the aid of the fluoroscope; the diagnosis as to the site of the pills depended on the fluoroscopic picture and not on the roentgenogram. The infants were observed generally at one hour intervals until the pills were noted to have left the stomach. In some cases, notably in a rather severe case of pyloric obstruction, referred to below, these fluoroscopic examinations necessitated frequent tests which had to be prolonged well into the night. It is a pleasure to thank Dr. Brim for

2. In order to ascertain whether our results were influenced mainly by the weight rather than the size of the pills, and whether this factor could account for the larger pill leaving the stomach as quickly or more quickly than the smaller one, we had some lighter pills made, composed of half licorice and half bismuth. Tests showed that these followed the same sequence in entering the intestine. In the summary of tests of Case 4 will be found one in which a lighter and a heavier No. 15 pill were given, and another in which a No. 9 and No. 15 licorice pill were given. In both instances the pills left the stomach within the same interval.

his cordial cooperation in this work, as well as Dr. Charles Gottlieb, who was kind enough to take the roentgenograms which illustrate this article. The fluoroscopic picture is almost always quite clear. The pill is sharply outlined within the infant's stomach, which, as is well known, is characterized by its large air bubble, which develops as a result of the lack of gastric peristole. When the infant is moved from one position to another, the pill rolls along the greater curvature in a corresponding direction. Sometimes it is at first difficult to decide whether it lies within the stomach or within the intestine. In such an event, the infant is turned first on one side and then on the other, and we note whether the pill moves, as should be expected, towards the pylorus, or towards the cardia. The crucial test consists in suspending the child by its feet and examining it in the inverted position. Under

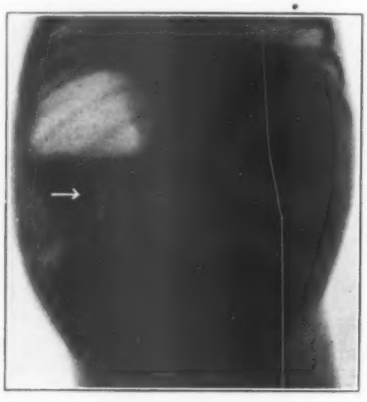

Fig. 7.—Upright posture. Pill at greater curvature; air bubbles and fluid above (exposure dorsoventral).

these conditions if the pill is within the stomach, it will assume a position close beneath the diaphragm, whereas if it is in the intestine, it will be lower down in the abdominal cavity. Figures 7 and 10 clearly demonstrate this point. The former shows the baby held in the normal vertical position and the pill fallen to the greater curvature; the latter shows the baby inverted, the pill having completely changed its position and dropped to the lesser curvature, the diaphragmatic portion of the stomach. In this position, surmounted by a layer of fluid and a large gastric air bubble, there can be no question as to whether the pill is within the stomach or the intestine.

Just as it is possible to alter the position of the pill in a vertical direction, so we can at will divert it laterally to the right or to the left. Figure 8 illustrates an infant lying on its right side and the pill at the pylorus; Figure 9 shows the same infant a few minutes later lying on its left side, and the pill far over along the left border of the

stomach. These pills were poorly made and show some disintegration within the stomach.

It may be stated as a corollary to this postural variation, that, merely by placing an infant on one side or the other, it was possible to hasten or to retard the passage of the pills into the intestine. A striking experiment of this kind is summarized under Case 4 (Table 4). It will be noted that the emptying time of the stomach of this infant was about one to two hours; that by keeping it on its left side (January 26 test), the passage of a small and a larger pill was delayed over four hours; that two days later, when the infant was placed on its right side, both pills had left the stomach within one and a quarter hours.

An opportunity presented itself of making use of this test in several cases of pylorospasm. The first was a typical case; a baby about 2

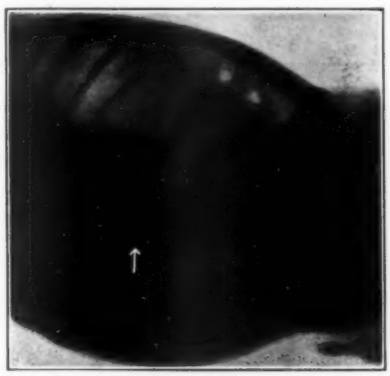

Fig. 8.—Same as Figure 7 five minutes later. Lying on right side. Pill at pylorus. Air bubble in left portion of stomach (exposure dorsoventral).

months old, breast fed, which had had projectile vomiting almost since birth. In the course of about five weeks some eighteen tests were carried out on this infant, comprising numerous fluoroscopic examinations and in some instances also roentgenograms. Visible peristalsis, obstruction to the duodenal catheter, marked gastric dilatation, and constant evidence of retention were present. The pills in this case were retained in the stomach frequently for about eight hours, and in one instance for over fourteen hours (Fig. 11). It seems unnecessary to consider the details of these several tests; however, the summary of Case 2, Table 2, may be of interest.

It may be well to mention a few points which Case 2 illustrates. In the first place it will be noted that the pills were very frequently macerated; evidently they were subjected to exceptional mechanical pressure within the stomach, incurred probably in the course of various

TABLE 2.—Time of Passage of Pills from Stomach in Case 2

William M., aged 2 months; pylorospasm; breastfed q. four hours.

Date	Size of Pills	Given at	Fluoroscoped	Site of Pills	Summary	Vomiting	Remarks
12/19/14	9, 15	9:45 a. m.	11:15 a. m.	Both in stomach	Macerated (both)	Projectile	Stomach large, distended.
			12:15 p. m.	Both in stomach			
			1:15	Both out of stomach	Out 2½-3½ hrs.		
12/20/14	9, 15	9:45 a. m.	11:00 a. m.	Both in stomach	Pills macerated	Projectile	
			12:00 m.	Both in stomach			
			1:00 p. m.	Both in stomach			
			1:30	Both out of stomach	Out 3¼-3¾ hrs.		
12/22/14	15, 21	10:00 a. m.	11:00 a. m.	Both in stomach	.....	Projectile	
			12:00 m.	Both in stomach			
			12:30	Both in stomach			
			1:15 p. m.	Both in stomach			
			2:00	Both in stomach			
			3:00	Both in stomach			
			4:00	Both in stomach			
			4:45	Both in stomach			
			5:15	Both in stomach			
			6:15	Both in stomach			
			7:00	Both out of stomach	Out 8¼-9 hrs.		
12/24/14	9, 15	10:00 a. m.	11:00 a. m.	Both in stomach	.....	Projectile	
			12:00 m.	Both in stomach			
			1:00 p. m.	Both in stomach			
			2:00	Both in stomach, fed			
			4:00	Both in stomach			
			5:30	Both in stomach			
			6:30	Both in stomach, fed			
			7:30	Both in stomach			
			8:45	Both out of stomach	Out 9½-10¼ hrs.		
12/27/14	9, 15	9:45 a. m.	11:15 a. m.	No. 9 out and No. 15 in stomach		Projectile	Papaverin, three doses (0.01 gm.). Fed at 2 p. m.
			12:15	No. 9 out and No. 15 in stomach			
			1:15 p. m.	No. 9 out and No. 15 in stomach			
			2:25	No. 9 out and No. 15 in stomach	No. 9 out within 1½ hrs.		
			3:30	No. 9 out and No 15 in stomach			
			4:30	No. 9 out and No. 15 in stomach	No. 15 out between 6¾-7½ hrs.		
			5:15	Both out of stomach			
12/28/14	9, 15	1:00 p. m.	2:15	Both in stomach	.....	Projectile Less	Papaverin, five doses (0.01 gm.). Fed q. four hours.
			3:15	Both in stomach			
			4:20	Both in stomach			
			5:20	Both in stomach			
			6:30	Both in stomach			
			8:00	Both in stomach			
			9:30	Both in stomach			
			10:40	Both in stomach			
			11:50	Both in stomach			
			12:50 m.	Both in stomach			
			1:30 a. m.	Both in stomach			
			2:15	Both in stomach			
			3:15	Both out of stomach	Out 13¼-14¼ hrs.		

12/30/14	9, 21	9:45 a. m.	11:30 a. m.	Both in stomach	Macerated	Very much diminished	Papaverin.
			12:15 m.	Both in stomach			
			1:15 p. m.	Both in stomach			
			2:30	Both in stomach			
			4:20	Both in stomach			
			5:30	Both in stomach			
			6:30	Portions out of stomach			
			7:15	All out of stomach	Out 8¾-9½ hrs.		
12/31/14	15, 21	9:45 a. m.	11:00 a. m.	Both in stomach	.....	Less	
			12:15 m.	Both in stomach			
			1:30 p. m.	Both in stomach			
			2:30	Both in stomach			
			3:30	Both in stomach			
			4:30	Both in stomach			
			5:30	Part out of stomach			
			6:00	Part out of stomach			
			7:15	Part out of stomach	Macerated		
			8:15	Part out of stomach			
			8:45	All out of stomach	Out 10½-11 hrs.		
1/4/15	15, Bismuth 15, Licorice	9:45 a. m.	11:00 a. m.	.....	.....	Once in last 4 days	Papaverin.
			12:30 m.				
			1:30 p. m.				
			2:30				
			3:30				
			4:30				
			5:30	Both out of stomach	Out 6¾-7¾ hrs.		
1/5/15	9 Licorice 15, Licorice	9:45 a. m.	11:00 a. m.	Both in stomach	.....	Less	Papaverin. Bottle and breast feeding.
			12:30 m.	Both in stomach			
			1:30 p. m.	Both in stomach			
			2:30	Both in stomach			
			3:30	Both in stomach			
			4:30	Both in stomach	Macerated		
			5:30	Both in stomach			
			6:30	Out of stomach	Out 7¾-8¾ hrs.		
1/9/15	9, 21	9:45 a. m.	11:00 a. m.	Both in stomach	.....	Less	Papaverin.
			12:00 m.	Both in stomach			
			1:00 p. m.	Both in stomach			
			2:30	Both in stomach			
			4:00	Both in stomach			
			5:00	Both in stomach			
			6:30	Both in stomach			
			7:30	Both in stomach	Macerated		
			8:30	Both out of stomach	Out 9¾-10¾ hrs.		
1/12/15	9, 15	9:30 a. m.	10:30 a. m.	Both in stomach	.....	None	Papaverin.
			11:30	Both in stomach			
			1:30 p. m.	Both in stomach			
			2:30	Both in stomach			
			3:30	Both in stomach			
			4:30	Both out of stomach	Out 6-7 hrs.		

TABLE 2.—TIME OF PASSAGE OF PILLS FROM STOMACH IN CASE 2—(*Continued*)

Date	Size of Pills	Given at	Fluoroscoped	Site of Pills	Summary	Vomiting	Remarks
1/14/15	9, 15	9:30 a. m.	10:30 a. m.	Both in stomach		None	Papaverin.
			11:30	Both in stomach			
			1:00 p. m.	Both in stomach			
			2:30	Both in stomach			
			3:30	Both in stomach			
			4:30	Both in stomach	Macerated		
			5:30	Both out of stomach	Out 7-8 hrs.		
1/15/15	9, 15	10:00 a. m.	11:00 a. m.	Both in stomach		None	Papaverin.
			12:00 m.	Both in stomach			
			1:00 p. m.	Both in stomach			
			2:00	Both in stomach			
			2:45	Both out of stomach	Out 4-4¾ hrs.		
1/16/15	9, 21	10:00 a. m.	11:00 a. m.	Both in stomach		Twice	No papaverin.
			12:30 m.	Both in stomach			
			1:30 p. m.	Both in stomach			
			2:30	Both in stomach			
			3:30	Both in stomach			
			4:30	Both in stomach			
			5:30	Both in stomach			
			6:30	Both in stomach	Macerated		
			7:30	Both out of stomach	Out 8½-9½ hrs.		
1/17/15	9, 21	10:00 a. m.	11:00 a. m.	Both in stomach		None	No papaverin.
			12:00 m.	Both in stomach			
			1:30 p. m.	Both in stomach			
			2:30	Both in stomach			
			3:30	Both in stomach			
			4:30	Both in stomach			
			5:30	Both in stomach			
			6:30	Both in stomach			
			7:30	Both in stomach	Macerated		
			8:30	Both out of stomach	Out 9½-10½ hrs.		
1/23/15	9, 21	10:00 a. m.	11:00 a. m.	Both in stomach		None	Adrenalin in IV (1-10,000).
			12:00 m.	Both in stomach			
			1:00 p. m.	Both in stomach			
			2:00	Both in stomach			
			3:00	Both in stomach			
			4:30	Both in stomach			
			5:30	Both out of stomach	Out 6½-7½ hrs.		
1/26/15	9, 15	10:00 a. m.	11:00 a. m.	Both in stomach			Adrenalin in IV.
			12:00 m.	Both in stomach			
			1:30 p. m.	Both in stomach			
			2:30	Both in stomach			
			3:30	Both in stomach			
			4:30	Both in stomach			
			5:30	Both out of stomach	Out 6½-7½ hrs.		

Epicrisis: Case of marked pylorospasm. Papaverin diminished the vomiting, although it did not seem to enable pills to enter intestine earlier.

attempts to pass the constricted pylorus. This case may also be of interest as affording repeated opportunity for testing the effect of papaverin on pylorospasm in an infant. This alkaloid is a derivative of opium and has been recommended especially to counteract the spasm of smooth muscle. It was given by us subcutaneously in the dose of 0.01 gm. Although it will be noted in the summary that the papaverin did not facilitate markedly the passage of the pills from the stomach, it had an undoubted effect on the vomiting, which became decidedly less marked under its influence. The papaverin delayed the passage of the pills through the intestine, as might be expected, considering its inhibiting effect on all smooth muscle.

In mild pylorospasm, however, papaverin may effect not only diminution of the vomiting, but hasten the passage of pills, and probably of food, from the stomach into the intestine. This was noted in

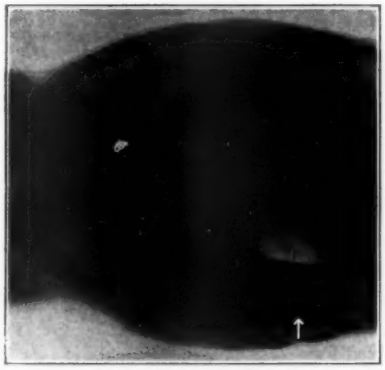

Fig. 9.—Same as Figure 8, five minutes later. Lying on left side. Pill at left border of stomach; air bubble extending to pyloric region (exposure dorso-ventral).

two instances. The first concerned a baby that had vomited since birth, and manifested obstruction to the passage of the duodenal catheter and a retention of the pills within the stomach for about seven hours (Case 3). When papaverin was given, both the small and the large pill were seen to have left the stomach after one or two hours. On discontinuing the drug, this period was extended to four to five hours, to be curtailed once more to one to two hours on resuming the medication. This case seemed particularly convincing as evidencing the value of this drug in some cases. Table 3 gives a summary of Case 3.

In the article on pylorospasm mentioned above, reference was made to a type of spasm of the pylorus termed "secondary pylorospasm." By this term an acute spasm of the pylorus is understood, a transitory hypertonic state. This condition is readily overlooked, as it is masked generally by the symptoms of the complicating dyspepsia.

TABLE 3.—Time of Passage of Pills from Stomach in Case 3

Sam H., aged 6 months; weight, 10¼ pounds.

Date	Size of Pills	Given at	Fluoroscoped	Site of Pills	Summary	Remarks
11/3/14	15, 21	2:00 p. m.	4:45 p. m.	Both in stomach		
			5:55	Both in stomach		
			6:40	Both in stomach		
			7:30	Both in stomach		
			8:20	Both in stomach		
			9:00	Both in stomach		
			9:30	Both out of stomach	Out 7-7½ hrs.	Vomited.
11/7/14	15, 21	10:00 a. m.	11:15 a. m.	Both out of stomach	Both out within 1¼ hrs.	Three doses papaverin (0.01 gm.).
11/12/14	9, 15	1:45 p. m.	3:55 p. m.	Both out of stomach	Out within 2⅛ hrs.	Five doses papaverin.
11/14/14	9, 15	10:00 a. m.	11:00 a. m.	Both in stomach	.....	Papaverin discontinued.
			12:00 m.	Both in stomach		
			1:00 p. m.	Both in stomach		
			2:00	Both in stomach		
			3:15	Both out of stomach	Out 4-5¼ hrs.	
11/24/14	9, 15	10:00 a. m.	11:00 a. m.	Both in stomach		
			12:15 m.	Both out of stomach	Out 1-2¼ hrs.	Three doses papaverin.
11/26/14	9, 15	10:10 a. m.	11:30 a. m.	Both in stomach		
			12:30 m.	Both in stomach		
			1:30 p. m.	Both in stomach		
			2:30	Both in stomach		
			4:00	Both in stomach		
			4:50	Both out of stomach	Out 6-6¾ hrs.	

Epicrisis: Case of mild pylorospasm. Infant had projectile vomiting almost since birth. Some obstruction to passage of catheter. Delay in passage of pills from stomach, the interval being shortened by means of papaverin from over sev en hours to about two hours. When the drug was discontinued the period was again prolonged (five hours).

A case of this nature was encountered in the fall of 1913 in one of the earliest tests made with the pills. The baby was 2 months old, and it had been possible to pass not only the ordinary duodenal catheter, but even a No. 18 and a No. 20, and the dilatable catheter (Case 5). We had concluded from the many tests made on this infant that its pylorus was especially patent and relaxed. Somewhat later it developed dyspepsia, manifested by marked vomiting. When the pills were

Fig. 10.—Same as Figure 9 five minutes later. Infant inverted in vertical position. Pill at diaphragm; air bubble at greater curvature (exposure dorso-ventral).

Fig. 11.—Case of pylorospasm (2 months old) showing No. 9 and No. 15 pills in stomach after thirteen hours. Notice marked dilatation of stomach and remnants in intestine of pills given on previous days.

introduced at this time, they did not leave the stomach for thirty hours, although in previous tests, the time of entering the intestine had been less than three or four hours. The case is summarized in Table 5.

In this connection we may add that tests by means of the pills have strengthened us in the opinion that in cases of pylorospasm there

TABLE 4.—Time of Emptying Stomach in Case 4
Morris F., aged 7 months.

Date	Size of Pills	Given at	Fluoroscoped	Site of Pills	Summary	Remarks
12/19/14	9, 15	9:45 a. m.	11:15 a. m.	Both out of stomach	Out within 1½ hrs.	Aspiration 1½ hours after feeding, showing 60 c.c. fluid in stomach.
12/22/14	9, 21	10:00 a. m.	11:00 a. m. 12:30 m.	Both in stomach Both out of stomach	Out 1-2½ hrs.	
12/30/14	9, 21	9:45 a. m.	11:30 a. m.	Both out of stomach	Out within 1¾ hrs.	
12/31/14	15, 21	9:45 a. m.	11:00 a. m. 12:15 m. 1:00 p. m.	Both in stomach Both in stomach Both out of stomach	Out 2½-3¼ hrs.	
1/4/15	15 15, Licorice	9:45 a. m.	11:00 a. m. 12:00 m.	Both in stomach Both out of stomach	Out 1¼-2¼ hrs.	Licorice pill used.
1/5/15	9, Licorice 15, Licorice	9:45 a. m.	11:00 a. m. 12:30 m.	Both in stomach Both out of stomach	Out 1¼-2¾ hrs.	Licorice pills used.
1/22/15	9, 15	10:00 a. m.	11:00 a. m.	Both out of stomach	Out within 1 hr.	Adrenalin m. V (1-10,000).
1/23/15	9, 15	10:00 a. m.	10:30 a. m. 11:00 11:30	Both in stomach Both in stomach Both out of stomach	Out 1-1½ hrs.	Adrenalin m. V (1-10,00).
1/26/15	9, 15	10:00 a. m.	11:00 a. m. 12:00 m. 1:30 p. m. 2:30 3:20	Both in stomach Both in stomach Both in stomach Both in stomach Both out of stomach	Out 4½-5½ hrs.	No drug. Kept on left side.
1/28/15	9, 15	10:15 a. m.	11:30 a. m.	Both out of stomach	Out within 1¼ hrs.	No drug. Kept on right side.

Epicrisis: Case with early emptying of the stomach. Passage into intestine markedly delayed by keeping infant on left side.

is frequently marked relaxation of the pylorus during the late stage of the illness. This phenomenon has already been noted in connection with a consideration of pylorospasm in its relation to duodenal catheterization. It was again clearly manifested by a case in which the pills throughout many tests had been greatly delayed in their passage from the stomach, but where six days previous to death, they were noted to have left the stomach within one to two hours. A sharp reversal of the fluoroscopic picture had taken place. These roentgenographic results harmonize with a similar change as to the passage of the catheter, and strengthen our opinion that pyloric relaxation is commonly a late manifestation of pylorospasm, and that it leads to a rapid passage of food into the intestine and to alimentary intoxication, the ultimate cause of death.

CONCLUSIONS

The foregoing study was carried out by means of keratin coated bismuth pills having definite circumferences corresponding to the size of the catheters, which we have shown can readily be introduced past the pylorus in infants. The purpose was to furnish a simple means of gauging the size of the pylorus and judging whether this sphincter was normally patent or not. It was found that under normal conditions objects do not leave the stomach in direct ratio to their size; that, in fact, larger objects are apt to be propelled into the intestine more quickly than smaller ones. This raises the question whether, following this analogy, food which has been insufficiently masticated may not remain in the stomach for a shorter rather than for a longer period than food that has been more thoroughly comminuted. Probably this is frequently the case. Under these conditions less work would be imposed on the stomach and proportionately more on the intestine, with the danger of consequent intestinal indigestion.

There was a marked difference in the emptying time of the stomach, according to the posture of the infant. The delay in the passage of the pills when the infant lay on its left side, and the hastening of their passage when the infant was placed on its right side, were almost constant phenomena. It would seem that this observation has clinical significance and is capable of practical adaptation. It may prove to be of advantage to place infants on the right side when there is an evident delay in gastric digestion.

In cases of pylorospasm there was a retardation in the passage of the pills from the stomach into the intestine. The degree of this delay varied in accordance with the degree of obstruction. The test does not enable us, however, to make a differential diagnosis as to the func-

TABLE 5.—Time of Emptying Stomach in Case 5

Anne R., aged 2 months; dyspepsia; weight, 8 pounds.

Date	Size of Pills	Given at	Fluoroscoped	Site of Pills	Summary	Remarks
11/26/14	.....	.....	.....	.....	.....	No. 15, No. 18 and No. 20 catheters passed into duodenum. Bile obtained.
11/29/14	.....	.....	.....	.....	.....	Same result as in first test.
12/2/14	.....	.....	.....	.....	.....	No. 15 and No. 18 catheters passed. Bile obtained. More difficult than previously. No. 20 could not pass. Grip—temperature 100.
12/4 & 5/14	.....	.....	.....	.....	.....	No. 15 and No. 18 catheters passed, No. 20 dilatable catheter passed; bile obtained.
12/9 & 11/14	.....	.....	.....	.....	.....	Larger dilatable No. 21 F. catheter passed.
12/21/14	.....	.....	.....	.....	.....	Balloon of catheter dilated to fully 0.5 cm., pulled back through cardia and pylorus.
12/24/14	15	11:00 a. m.	3:30 p. m.	Out of stomach	Out within 4½ hrs.	Only one examination made.
12/26/14	15	11:00 a. m.	2:00 p. m.	Out of stomach	Out within 3 hrs.	Only one examination made.
12/29/14	15	12:00 m.	3 hours	In stomach	.....	Tried to pass duodenal catheter fully six times without success. No bile obtained. Grip. Slight vomiting.
			4 hours	In stomach		
			5¼ hours	In stomach		
			11 hours	In stomach		
			24 hours	In stomach		
			27¼ hours	In stomach		
			29 hours	In stomach		
			30 hours	In stomach	In stomach at least 30 hrs.	
			48 hours	Out of stomach		

Epicrisis: Case with marked relaxation of pylorus, as shown by the rapid passage of pills and frequent passage of catheters of large diameter. Secondary pylorospasm manifested by failure to pass catheter and long delay in the passage of pill into the intestine.

tional or the organic nature of the obstruction. In cases characterized by pyloric obstruction it was found that the pills left the stomach according to their size. In mild cases of spasm papaverin was found to be effective in definitely shortening the time of exit of the pills; in severe cases it lessened vomiting but did not facilitate the passage from the stomach.

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THE FREQUENCY OF INFECTION WITH THE TUBERCLE BACILLUS IN CHILDHOOD*

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A statement in one form or another that 90 per cent. or more of children are infected with the tubercle bacillus by their fourteenth year has frequently appeared in both the medical literature and the lay press of the last few years. This is one of the factors which has been urged in support of the theory that the pulmonary tuberculosis of early adult life is a lighting up of an old latent infection acquired during childhood—a possible but as yet unproved hypothesis. In tracing back the authority for this statement we find that it is almost entirely based on the figures of Hamburger and Monti,[1] who made tuberculin tests on 532 children convalescing from diphtheria and scarlet fever in the wards of a public hospital in Vienna. These children were first tested by the cutaneous method, and if the reaction was negative, 1/100 milligram, or more, of tuberculin was injected and the subcutaneous local (*Stich*) reaction recorded. Positive tuberculin tests are regarded as evidence of infection with the tubercle bacillus, whether the tuberculosis be active, latent or healed. The detailed results of their tests are shown in Table 1.

TABLE 1.—CHILDREN SUBMITTED TO TUBERCULIN TEST (HAMBURGER AND MONTI)

Age (Years)	No. Children	Positive Reactions	Per Cent. Positive
Under 1	23	0	0
1-2	46	4	9
2-3	56	11	20
3-4	75	24	32
4-5	50	26	52
5-6	63	32	51
6-7	46	28	61
7-8	30	22	73
8-9	35	25	71
9-10	26	22	85
10-11	29	27	93
11-12	19	18	95
12-13	17	16	94
13-14	17	16	94

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* Submitted for publication, April 5, 1915.

1. Hamburger and Monti: München med. Wchnschr., 1909, lvi, 449.

No one else has reported such a high incidence of infection among children free from clinical tuberculosis. In fact, von Pirquet,[2] to whom credit must be given for first showing the frequency of infection with the tubercle bacillus in childhood, obtained lower figures among the same class of children in Vienna even when cases of clinical tuberculosis were included in his statistics. Von Pirquet used only the cutaneous test in determining the presence of an infection. In Table 2 his figures are given in a condensed form for various age periods. In the second part of the table the figures give the percentage of positive reactions in children free from clinical manifestations of tuberculosis.

TABLE 2.—RESULTS OF CUTANEOUS TESTS (VON PIRQUET)

	Including Cases of Tuberculosis			Excluding Cases of Tuberculosis		
Age (Years)	Total No.	Positive Reactions	Per Cent. Positive	Total No.	Positive Reactions	Per Cent. Positive
Under 1	410	21	5.	388	0	0.
1-2	116	24	20.	89	0	0.
2-4	208	74	35.	162	22	13.
4-7	264	127	48.	189	53	30.
7-10	216	138	64.	154	74	48.
10-14	193	155	80.	147	105	70.

The figures of both Hamburger and von Pirquet were obtained from tests made on children of the poorer classes of society in Vienna —a city where tuberculosis is notoriously frequent—and who were living in an environment which is only found in the slums of the older continental cities. As von Pirquet said, "In other cities it (the percentage) will hardly be as high . . ."[3]

In the course of our work at the St. Louis Children's Hospital we gained the impression that positive tuberculin reactions were much less frequent than might be expected from the general idea which prevails, although we were dealing with children of relatively the same class as Hamburger, as our patients are all from the poorer and more congested districts of St. Louis. In order to see whether this was an impression or a fact, the clinical records of the past three years were carefully gone over and the results of the tuberculin tests tabulated. As our results gave figures so different from the general impression of the frequency of infection, and as we have been unable to find any reports of large series of tests on children in American cities, it has seemed of sufficient interest to place our statistics on record. Our figures do not represent a special study of the frequency of tuberculin reactions in childhood, but the results of tests made for diagnostic purposes on hospital patients regardless of the cause for their admission. Tuberculin tests are made on all infants under two

2. Von Pirquet: Jour. Am. Med. Assn., 1909, lii, 675.
3. Von Pirquet: Trans. VI Internat. Cong. Tuberc., 1908, ii, 559.

years admitted to the hospital as a part of the routine examination, but the tests in older children do not represent consecutive admissions. As a rule tests are only made in children over two years when there is some indication. In Table 3 the figures include all cases, and in Table 4 the cases with *clinical manifestations of tuberculosis have been deducted*. As all of these children were admitted to the hospital for some pathologic condition, it is very probable that the figures in Table 4 are higher than would be obtained if an examination of "normal school children" of the same social conditions were made. The cutaneous test of von Pirquet was used in all cases. Full strength "old tuberculin" was used and the scarification made through drops by means of a von Pirquet scarifier. Whenever a reaction was questionable a second test was made. The activity of the tuberculin was frequently checked by tests on known cases of tuberculosis which had previously given positive reactions. As the children were in the hospital no difficulty was experienced in observing and recording the reaction at various intervals after the test was applied. For the last six months we have followed negative cutaneous reactions with intradermic injections of 1/100 and 1/10 mg. of tuberculin, as the cutaneous test is regarded by Hamburger as only being positive when the individual has a degree of sensitiveness which reacts to 1/1,000 of a mg., or less, injected intradermally.[4] Hamburger contends that the subcutaneous local and the intradermal reactions are from 25 to 30 per cent. more delicate than the cutaneous test of von Pirquet, but in 263 cases tested in this way the increased percentage of positive reactions was only 10 per cent. and not 25 to 30 as Hamburger found. We may therefore regard these children in our series tested by the cutaneous method alone as giving a very fair estimate of the extent of infection with the tubercle bacillus among the children of the lowest strata of society in St. Louis. The fact that the children in our series were hospital patients accounts for the large amount of clinical tuberculosis. This is especially the case for the figures for the first year of life.

TABLE 3.—TOTAL REACTIONS INCLUDING CASES OF CLINICAL TUBERCULOSIS

Age (Years)	No. Children Tested	Positive Reactions	Per Cent. Positive
Under 1	224	25	11
1-2	137	34	24
2-4	188	57	30
4-6	206	68	33
6-8	182	74	40
8-10	157	69	44
10-12	115	45	40
12-14	112	54	48
	1321	426	

4. Hamburger: Die Tuberkulose des Kindesalters, 1912, p. 62.

TABLE 4.—REACTIONS IN CHILDREN WITHOUT CLINICAL MANIFESTATIONS OF TUBERCULOSIS

Age (Years)	No. Children Tested	Positive Reactions	Per Cent. Positive
Under 1	202	3	1.5
1-2	109	6	5.5
2-4	163	32	19
4-6	172	40	23
6-8	152	44	29
8-10	126	38	30
10-12	107	37	34
12-14	94	36	38
	1125		

From these tables it will be seen that the percentage of positive tuberculin reactions among children of relatively the same social conditions was much lower in St. Louis than in Vienna. For purposes of comparison we have grouped the percentages of positive reactions into age periods in Table 5. These figures are for children without clinical tuberculosis.

TABLE 5.—PERCENTAGE OF POSITIVE REACTIONS GROUPED BY AGE PERIODS

Age (Years)	St. Louis	Vienna	
		von Pirquet	Hamburger
Under 1	1.5	0	0
1-2	5.5	0	9
2-4	19	13	26
4-7	25	30	54
7-10	30	48	75
10-14	36	70	94

In St. Louis the increase after the 2 to 4 year age period is relatively slow, while in Vienna this corresponds to the period of most rapid increase.

In a cursory search of the literature we were surprised to find the few series of cases reported in view of the importance and publicity that has been given to the figures of Hamburger. In America Shaw and Laird[5] tested 330 institutional children and found a maximum of 45 per cent. giving positive reactions between the ages of 6 and 14. Wachenheim[6] obtained but 5 positive reactions among 80 non-tuberculous children from the congested districts of New York. Several larger series of tests among infants and young children have been reported. We have found a number of reports from European cities and it is noticeable that among these Hamburger's figures give the highest percentage of positive reactions. Øverland[7] tested 843 school children in Bergen and found the positive reactions reached a maximum of 50 per cent. at the 12 to 14 year age period. In nearby rural

5. Shaw and Laird: Trans. Nat. Soc. Study and Prev. Tuberc., 1909, v, 350.
6. Wachenheim: AM. JOUR. DIS. CHILD., 1913, v, 466.
7. Øverland: Abstr. in Internat. Centralb. f. Tuberk., 1914, viii, 818.

districts the incidence of infection was considerably less. Stawsky[8] found the infections reached about 60 per cent. between the ages of 12 and 16 years in Odessa. Amenta[9] in Palermo tested 800 children under 12 years by the cutaneous method and found a rather higher incidence: 0-6 months 17.5 per cent.; 6-12 months 30 per cent.; 1-2 years 28 per cent.; 2-4 years 47.5 per cent.; 4-6 years 72 per cent.; 6-8 years 75 per cent.; 8-12 years 83 per cent. Calmette, Grysez and Letulle[10] reported tests on 1,226 children in Lille and found positive reactions in 81 per cent. between the ages of 5 and 15.

It is thus seen that there is no uniformity in the figures obtained in different countries and cities, a condition which can hardly be attributed to faulty or differences in technic with such a simple procedure as the cutaneous tuberculin test. As a matter of fact, one would hardly expect to obtain uniform figures, as the incidence of tuberculosis is such an accurate index of social and living conditions, and these factors differ so widely in different communities. For example, the death rate for all forms of tuberculosis per 100,000 of population in St. Louis in 1910 was 184, while in Vienna the death rate for pulmonary tuberculosis alone was 249 per 100,000. That the extent of infection in childhood bears a relationship to the degree of exposure to clinical tuberculosis has been shown by Sachs and others, and more recently by Lamson. Lamson[11] found that 67 per cent. of individuals in families in which there was an open case of tuberculosis showed evidences of infection with the tubercle bacillus. In families in which there was "latent" tuberculosis, 22 per cent. showed evidences of infection, and in families in which there was no tuberculosis, only 2½ per cent. We have been unable to find a report of a series of tuberculin tests on children of families living in the better residential districts of our large cities, but there is every reason to believe that the extent of the infection is much less than is found in the congested districts. The extent of the infection with the tubercle bacillus in childhood is in all probability directly proportionate to the amount of tuberculosis in a given community, and to the degree of intelligence to which people are educated regarding the dangers of, and the method of preventing, the spread of tuberculosis.

It is quite possible that the extent of infection among certain classes may reach 90 per cent. by the fourteenth year, but this is extreme, and most statistics give much lower figures. It must be kept in mind that all of the figures which have so far been published are from hospital or dispensary children. It would be well worth while if some one

8. Stawsky: Abstr. in Internat. Centralb. f. Tuberk., 1914, viii, 821.
9. Amenta: Abstr. in Internat. Centralb. f. Tuberk., 1914, viii, 632.
10. Calmette, Grysez and Letulle: Presse méd. 1911, xix, 651.
11. Lamson: Studies in Public Health, Univ. of Minnesota, 1913.

with an extensive private practice among the better classes of society would publish the results of a large series of tests. It would seem, however, that although infection with the tubercle bacillus is widespread, the usual statements as to the extent of the infection as a whole are gross exaggerations.

Although not strictly germane to the subject of this report, it may not be amiss to express the opinion we have formed in the course of our work in regard to the value of the tuberculin test as a diagnostic measure in childhood. Positive reactions in early childhood and infancy are generally regarded as of value. With this we concur. The statement is then usually made that the tests are of little or no value in older childhood (from 3 to 7 years on, according to various observers) as the latent infections are so numerous by this time. From our figures it will be seen that latent infections are not so common in St. Louis as has been inferred from the statistics from other cities. As far as negative reactions in clinical cases of tuberculosis are concerned, we have met with but six cases of tuberculin insensitiveness. Five of these were in children with high temperatures, tested shortly before death occurred, and the sixth was a case of old, long-standing abdominal tuberculosis in which a mesenteric gland removed at operation showed tubercles on histologic examination. In this connection it is interesting to note that several cases of "hip joint" disease on the orthopedic service of Dr. Allison, which were at first regarded as tuberculous, but which gave repeated negative tuberculin tests, subsequently proved to be cases of osteochondritis juvenilis (Perthes disease). Even if latent infections are frequent enough to lessen the diagnostic value of positive tuberculin reactions, they have not infrequently given support to the data obtained on physical examination in the diagnosis of a tuberculous process. Furthermore, we are of the opinion that sufficient emphasis has not been placed on *negative* tuberculin tests in older childhood, particularly in children with pulmonary lesions. The clinical similarity between pulmonary tuberculosis and chronic influenzal and pneumococcic processes in older children is very marked, and in the diagnosis of these conditions we have found negative tuberculin reactions of great aid.

SUMMARY

A study of tuberculin tests in 1,321 hospital children in St. Louis shows that the percentage of positive reactions reaches a maximum of 44 per cent. at the age period of 10 to 14 years, including cases with clinical tuberculosis. If children with clinical tuberculosis are excluded the percentage giving positive reactions at the 10 to 14 age period is only 36. These figures are much lower than the usual "90 per cent."

figure for the incidence of infection with the tubercle bacillus in children by their fourteenth year, which has gained such widespread publicity and which is based on the figures of Hamburger for Vienna.

The extent of infection among children varies in different cities and countries and is dependent on such factors as living and social conditions, the amount of tuberculosis in the community, the exposure of the child to open tuberculosis, and in all probability varies among different classes of society in the same community. No conclusions as to the extent of infection with the tubercle bacillus can be drawn from the statistics of any one city or class, and the statement that "90 per cent. or more" of individuals are infected by puberty is an extreme exaggeration of the actual conditions which exist.

The intradermic tuberculin test has given but a slight increase in the percentage of positive reactions over the percentage of positive cutaneous reactions. We have found that positive tuberculin reactions are of much service in the diagnosis of tuberculosis in infancy and early childhood, and that both positive and negative reactions in conjunction with the physical signs and symptoms have been of diagnostic value in individual cases among older children, although as a whole they do not indicate whether the infection is active or latent.

A CASE OF BILATERAL GLIOMA OF THE RETINA*

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The facts concerning the origin, course and prognosis of the non-pigmented cancer of the retina occurring in children are well known. Wintersteiner's classical monograph, in which he describes 467 cases, has been referred to as a basis of their remarks by authors who have published accounts of individual cases. Wehrli[1] believes that retinal hemorrhages are the prime etiologic factor, because he has found rosettes, the centers of which were formed by the smallest vessels. These vessels were fully developed and for the most part degenerated, while their lumen contained remnants of red blood corpuscles surrounded by epithelial-like tumor cells.[2] Lindenfeld[3] in the study of the normal eyes of the fetus speaks of "granular rosette folds and folds of undifferential cells. From the cells may be derived the similarly arranged cells of retinal glioma." Fuchs'[4] description of the pathology and changes voices the generally accepted opinion:

> It develops ordinarily from two granular layers of the retina. The tumor is composed of small, cylindrical, epithelial cells and a soft basement substance. In many cases the cells are arranged about an open space, like the cross section of a gland tubule. The multiplication of the tumor cells takes place mainly in the immediate vicinity of the numerous wide blood vessels. Here are the youngest cells which displace the older cells, the latter undergoing necrosis. Neoplasm germs pass from the degenerated retina into the choroid and vitreous, where they subsequently develop into small independent nodules.

In considering the etiology, heredity should be given an important place. Many are the instances recorded in which the disease has manifested itself in the parents and children, or in several children of the same parents. Fuchs[4] says, "A congenital morbid disposition very often lies at the bottom of glioma" and illustrates his statement by the following history: a 4 year old boy with advanced and perforating glioma of the right eye; a 2 year old brother with glioma of the right eye; finally a child of the same parents a few months old, with typical congenital coloboma of the iris and choroid. De Gouva[5]

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* Read before the Section on Ophthalmology, College of Physicians, Philadelphia, March 18, 1915.

1. Wehrli: Graefe's Arch. f. Ophthal., 1909, p. 504.
2. Ophth. Year Book, 1910.
3. Lindenfeld: Klin. Monatsb. f. Augenh., April, 1913.
4. Fuchs: Text Book, edited by Duane.
5. De Gouva: On the Heredity of Glioma of the Retina, Ann. d. Oculist, January, 1910, cxliii.

removed the right eye of a 2 year old boy for glioma in 1872. There was no recurrence, and on growing up he married a woman in whose family there was no tendency to the formation of neoplasm. They had seven children, the second and third of whom, both girls, died of double retinal glioma. Von Hoffman, Jr.,[6] and Owens[7] recorded other instances.

The list of affections which may be confounded with glioma and, which are usually classified under the unexpressive and negative term of "pseudoglioma," is appallingly large, and the diagnostician who endeavors to be accurate and specific cannot be satisfied with the two classes of glioma and pseudoglioma. Curtil[8] enumerates the affections to be differentiated: metastatic ophthalmia, especially in the form of exudative choroiditis; metastatic or traumatic exudative or suppurative lesions of the vitreous; choroidal tuberculosis; simple detachment of the retina; certain congenital anomalies; cysticercus of the deep membranes of the eye.

It is not enough to say prior to dismissing the subject of differentiation that the history and clinical symptoms of true glioma would indicate the diagnosis. While this may be true in some of the diseases mentioned, it is not true for all. Simple retinal detachment, for example, rarely occurs spontaneously. Malignant myopia, traumatism or constitutional affections, usually point to the etiology. Moreover, the separated retina is wavy, its arteries and veins of the same dark color and readily seen, and when transillumination may be employed, throws no dark shadow. Metastatic choroiditis has a distinct history, occurs at any age, is rapidly progressive, and has a dark yellow or reddish color and not the almost pink white of glioma. Advanced choroidal tuberculosis is almost never found in very young children and may be regarded as a local sign of general tuberculosis.

Congenital anomalies remain unchanged in their appearance for many years. Cysticercus may be diagnosed in most instances with the ophthalmoscope. Friedenwald[9] and de Schweinitz[10] cite cases of "retinitis with massive exudation" that resemble glioma in their ophthalmoscopic appearance. They are essentially different in their etiology, history and course and will not be confused by the thoughtful observer. In a case recorded by Krauss[11] and in others to which he refers in his paper, the diagnosis is more difficult. A boy of 8 received an injury to the eye at birth. Seven months before examination, and

6. Von Hoffman, Jr.: Heidelberg Congress, 1908.
7. Owens: Royal London Ophth. Hosp. Rep., 1905.
8. Curtil: Rev. gén. d'ophtal., Dec. 31, 1910.
9. Friedenwald: Trans. Am. Ophth. Soc., 1914, xii, Part iii.
10. de Schweinitz: Trans. Am. Ophth. Soc., 1914, xii, Part i.
11. Krauss: Ophthalmology, April, 1913.

several times since, there had been signs of moderate inflammation. The pupil was dilated and fixed, anterior chamber shallow and a few posterior synechiae were present. A greenish-yellow reflex shone from behind the lens on the temporal side, and later, all around the pupil. The eye was enucleated. There was total retinal detachment, subretinal hemorrhages and a mass of partly organized inflammatory exudate between the retina and the lens. The blood vessels of the retina were degenerated but the iris, choroid and sclera were practically normal. I believe his case belongs to a special group properly called "pseudoglioma" which originates primarily in the retina. I agree with those who hold that there is a distinct disease of the retina occurring in children which produces clinical symptoms closely simulating glioma and is of retinal and not choroidal origin. Even in Krauss' case real glioma might be excluded before enucleation on account of the age of the patient, the recurring attacks of inflammation and the absence of the signs of secondary glaucoma.

Devereux Marshall states that the statistics of the Moorfields Hospital show that about 60 per cent. of the cases of glioma occur in the first two years of life and that about 80 per cent. occur during the first three years. He studied thirty-two cases; of these there were fifteen which had exceeded the three year limit, the time running from three years and four months to six years and nine months. Wintersteiner found 75 per cent. within the first three years. Curt Adam[12] studied the records of forty-seven cases. All the patients were less than 12 years old, 94 per cent. being under 4 years. The attacks of inflammation, the posterior synechiae and the yellowish-green foreign material in the posterior chamber, and the absence of secondary glaucoma, point to retinal or choroidal inflammation rather than a glioma.

CASE REPORT

History.—Boy 2 years old. Parents healthy. No family history of cancer or other forms of tumor or of congenital affections of the eyes. The patient has a sister two years older than himself who is apparently well and strong. One year ago a "shine" was noticed in the pupil of the left eye. At present the eyeball is larger than its fellow, the pupil dilated and immobile, anterior chamber shallow, lens clear and the entire vitreous occupied by a shining, white, irregular mass, traversed by a few blood vessels. Tension +1. No perception of light. Under chloroform anesthesia, and through the pupil dilated with homatropin, a small, round, white mass projecting several diopters into the vitreous and situated close to the nerve head on its temporal side was seen in the *right* eye. An object held in the center of the field of fixation was not seen by the child, but even small objects were readily perceived in the periphery. The left eye was enucleated. The pathologist's report is as follows:

Pathologist's Report.—The specimen is that of an eyeball with attached portion of optic nerve. It measures 2.8 by 2.1 cm. and weighs 3.8 gm. The eyeball is soft and the anterior chamber is partly collapsed. There is no apparent

12. Adam, Curt: Ztschr. f. Augenh., 1911, xxv, 330.

external evidence of disease, except for a marked grayish opacity of the lens. On section, after hardening the eyeball, an irregularly outlined, grayish, soft tumor mass occupies the vitreous, apparently arising near the entrance of the optic nerve and extending into the vitreous to within a few millimeters of the lens. This mass measures 1.4 by 0.9 cm. in its greatest diameters. The retina everywhere appears to be intact.

Histology: The sections show the tumor mass apparently arising from the granular layers of the retina, and made up of numerous small round cells with deeply staining nuclei lying amongst a fairly abundant network of neuroglia fibers. The blood vessels, with well formed walls and filled with red cells, appear directly amongst the cellular elements. The tumor growth has not involved the optic nerve.

Diagnosis: Glioma of retina.

Subsequent Course.—Three weeks later the left orbit was fitted with an artificial eye. The tumor in the right eye had increased in size and vision was measurably reduced. It was my distasteful duty to inform the parents that enucleation of the right eye must be performed as soon as vision should be lost, because by this means only was there a chance of saving the child's life.

My situation was exactly that described by Jacqueau[13] and doubtless experienced by all who have had cases of bilateral glioma to deal with. He emphasized the tragic dilemma of the surgeon who is called on to render a decision in such cases. Van Duyse[14] in discussing Gallemaert's case of double glioma says that even in such instances the patient may survive if enucleation has not been delayed too long. He referred to a case of his own in which fourteen months had elapsed without recurrence since enucleation of the second eye, which had been removed two years after the first.

Postponement of enucleation is fatal. Once the diagnosis is established, sentimental objection by parents, and alas! sometimes by the family physician, should be given no serious consideration. The disease will surely spread, either by rupture of the sclera and extension to the orbital contents, or posteriorly along the nerve, and then enucleation or even evisceration would be useless. Spontaneous cure cannot be expected, notwithstanding Lindenfeld's[15] experience, who reports a case of spontaneous cure, the third of its kind to be supported by pathologic findings. He saw a 4 year old child with fungoid glioma of the right eye and a shrunken left globe. Both eyes were enucleated. The clinical diagnosis of bilateral glioma was confirmed histologically only as regards one eye, since no trace of glioma could be demonstrated in the shrunken eye. But the belief that this eye had previously suffered from the same condition was established by the entire clinical history and course of the disease. The "spontaneous cure" was due to complete necrosis of the tumor. This "pathologic cure" is to my mind even worse than enucleation.

13. Jacqueau: Clin. Ophth., 1912, xix, 733.
14. Van Duyse: Soc. Belge d'ophth., 1911, xxv, 19.
15. Lindenfeld: Graefe's Arch. f. Ophthal., 1912, lxxxvi, 141.

In the bilateral cases, which constitute about 25 per cent. of the total number, the disease begins in one eye and the second eye is involved some months later. The question arises whether prompt enucleation would save the second eye. In other words, could the second eye become involved by sympathy?

In this connection the four cases reported by Ayers[16] are of interest: 1. Enucleation was performed when the tumor had advanced to the cornea and quite filled the globe. The patient is now 14 years of age and in good health. 2. The eye was enucleated after blindness of several months. "This child is now 8 years old and in excellent health." 3. The tumor was almost in contact with the lens, the pupil dilated, and T+1. The child died two weeks after enucleation from extension of the growth into the brain. 4. Enucleation. "The child is now 2½ years old and bids fair to pass the three years' limit of immunity."

These histories emphatically negative the supposition that the sound eye is involved sympathetically.

Glioma may be briefly described as a malignant tumor arising from the granular layers of the retina, almost pure white, traversed by a few blood vessels, steadily advancing, without pain or symptom of constitutional irritation, until it has reached its second or glaucomatous stage. Unless the eyeball is enucleated, the tumor will perforate the coats of the eye and invade the orbit, or it will travel backward in the optic nerve and invade the brain. The only conservative treatment is the enucleation of the eye at the earliest moment. By this means the second eye may not be involved and the life saved. De Kleijn[17] states concerning four cases in which the optic nerve was microscopically free from disease at the time of operation, that the subsequent records show no fatalities. When the optic nerve was affected but not up to the point of division, two patients had been well for eight and seven years, respectively, and four had died. Of five cases in which the optic nerve was affected at the point of section, only one patient had lived, the interval since operation being four years. The three-year limit is generally accepted. Statistics show that fatalities occur as a rule within the first year, and should the patient survive without recurrence until the termination of the third year, the prognosis is favorable.

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16. Ayers: Arch. Ophthal., July, 1906.
17. De Kleijn: Arch. f. Ophthal. (Graefe's), 1911, lxxx, 371.

CLOTHING AS A FACTOR IN THE PRODUCTION OF HEAT STASIS*

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The studies of the relation of heat to the high infant mortality of summer have not yielded uniform results. A more careful review of the procedures which have been employed in these studies readily explains these contradictory conclusions. The external factors, such as food, high outdoor and indoor temperatures, circulation of air, etc., have received most of the attention; while the most important internal factor, i. e., the heat regulatory power of the infant, has been neglected by most authors. The question, then, which confronts us is: the reaction of the infant to the changes in its environment, with special reference to its heat regulation.

Heat equilibrium of the organism depends on the proper balance between:

I. Heat production,

II. Heat elimination.

I. Heat production is influenced chiefly by:

1. Food,
2. Work.

1. That food can influence heat production is shown by Rubner,[1] who was able to increase the heat production of a dog 44 per cent. by excessive feeding. A food, the caloric value of which meets the requirements of the organism at a moderate temperature, may become excessive in summer if the heat loss is interfered with. This may be further emphasized by the composition of the food.

2. That muscular activity increases heat production has been very well known, for instance, by Howland's[2] calorimetric observations on infants. He has shown that the heat production of infants, crying or making active movements, is considerably greater than that of those asleep.

II. Heat Elimination. Heat loss is dependent almost entirely on:

1. Radiation.
2. Conduction.
3. Evaporation.

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* From the Otho S. A. Sprague Memorial Institute Laboratory of the Children's Memorial Hospital.

1. Rubner: Lehrbuch der Hygiene, 1907. Ed. 8.

2. Howland: The Scientific Basis for the Artificial Feeding of Infants, AM. JOUR. DIS. CHILD., 1913, v, 390.

1 and 2. Loss by radiation and conduction is proportional to the difference between the surface temperature of the body and that of the surrounding air. As the temperature of the air approaches that of the body surface, the loss due to these two factors becomes less. It is zero at temperatures equal to or above that of the body surface.

Good circulation of air aids heat loss by conduction and radiation, by a more frequent exchange of air strata about the individual. The surface temperature of the clothing is less than that of the uncovered body and, therefore, approaches more nearly that of the external air. This fact accounts for the less rapid loss of heat through radiation and conduction in clothed individuals.

3. Evaporation gains in importance as a means of heat loss at high temperatures, coincident with the appearance of visible perspiration, and becomes the principal means of heat loss when the temperature of the surrounding air is higher than that of the body. Evaporation is increased by good circulation of the air and inhibited by high humidity. One must not lose sight of the fact, however, that water is constantly lost through the skin independently of the action or presence of sweat glands. This has been clearly shown by Loewy[3] in a recent publication, in which he reports observations on the loss of water from the skin in three individuals who had a peculiar ectodermal anomaly, in that the sweat glands, and to some extent the sebaceous glands, were absent. At moderate temperatures, with the body at rest, the loss of water from the skin of these individuals was as great as that from the surface of normal individuals. From this he considers the so-called insensible perspiration a physical, not a physiologic phenomenon. The work of Galeoti and Macri,[4] although their method is not free from objection, seems to add some support to this view. They found that the loss of water by insensible perspiration on the different portions of the skin of the same normal individual is not proportional to the number of sweat glands present there.

Mendelssohn[5] studied heat regulation of the infant and found this mechanism to be more efficient at high than at low surrounding temperatures. He also emphasizes the importance of heat loss by evaporation from the skin and lungs at high temperatures (e. g. 40 C.). He calls attention to the increased respiratory rate at these temperatures and states that at such temperatures heat loss by radiation and con-

3. Loewy: Untersuchungen über die physikalische Hautwasserabgabe, Biochem. Ztschr., 1914, lxvii, 4, 5, p. 243.

4. Galeoti and Macri: Ueber die Perspiratio Insensibilis unter normalen und pathologischen Bedingungen, Biochem. Ztschr., 1914, lxvii, 6, p. 472.

5. Mendelssohn: Ueber das Wärmeregulationsvermögen des Säuglings, Ztschr. f. Kinderh., 1913, v, *269*; Beobachtungen über Hauttemperaturen der Säuglinge, Ztschr. f. Kinderh., 1912, iii, 292.

duction is relatively unimportant. He states, however, that in the majority of infants no sensible perspiration was noted. He mentions the prophylactic value of cool surroundings and scant clothing during hot weather. Rubner had previously shown the importance of heat loss by evaporation from the skin and lungs at high temperatures.

Rietschel,[6] Kleinschmidt,[7] Liefmann and Lindemann[8] and others, and more recently Helmholz,[9] have studied the relation of high indoor and outdoor temperatures to infant mortality. Helmholz made careful observations on forty-six infants, extending over a period of eight summer weeks. These infants attended infant welfare stations in Chicago, and were considered fairly normal tenement babies. Their feedings were supervised by the infant welfare physicians, who also gave advice concerning hygiene. A nurse visited the homes each day. Rectal temperatures and maximal and minimal room temperatures were recorded daily. Notes were also made on the stools and general condition of the infants. Helmholz's observations show no definite relationship between individual high room temperature readings and individual cases of hyperthermia and gastro-intestinal disturbance. He says: "The direct effect of heat alone must be considered of relatively little importance. Infants can tolerate temperatures decidedly above those usually supposed to be harmful, without any bad effects; in fact, can gain in weight during such time." He points out that in his cases heat, humidity and diet were quite uniform and that certain infants showed ill effects even at relatively low temperatures, a fact which requires the investigation of other factors allowing greater individual variability. "Such a factor we have in clothing."

This work of Helmholz and the fact that food, temperature, humidity and ventilation have been investigated to a considerable extent, has led us to undertake an experimental study of the effect of clothing—a factor which has received relatively little attention, and which can, in various ways, influence heat loss.

EXPERIMENTAL PROCEDURE

In each of eight experiments two puppies of the same litter, ranging in age from 4 to 6 weeks, were put into an incubator in which was maintained a temperature simulating that of a hot summer. A jacket was placed on one of the puppies in the incubator; the other remained uncovered. In six of the experiments an additional control puppy wearing a jacket was kept at room temper-

6. Rietschel: Sommerhitze, Wohnungstemperatur und Säuglingssterblichkeit, Ztschr. f. Kinderh., 1911, i, 546.

7. Kleinschmidt: Der Einfluss der Hitze auf den Säuglingsorganismus, Monatschr. f. Kinderh., 1910-11, ix, 455.

8. Liefmann and Lindemann: Der Einfluss der Hitze und die Sterblichkeit der Säuglinge in Berlin, etc., Vierteljahrb. f. öff. Gesundheitspflg, 1911, xviii, 333.

9. Helmholz: The Relation of Heat to the Morbidity and Mortality of Infants from Gastro-Intestinal Diseases, Jour. Am. Med. Assn., 1914, lxiii, 1511.

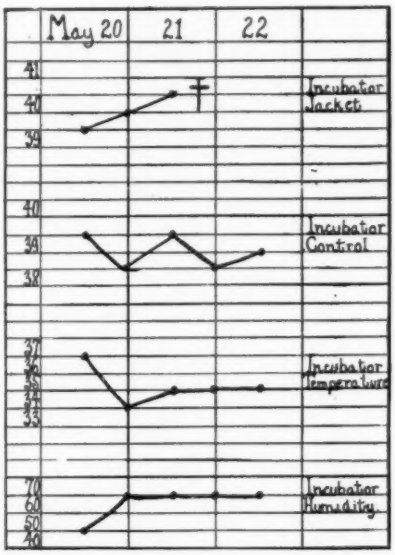

Fig. 1.—(Experiment 1.) Rectal temperatures of incubator puppies (jacketed and control), the incubator temperature and humidity, etc. The plus sign indicates the death of the animal. This explanation applies to all of the following charts.

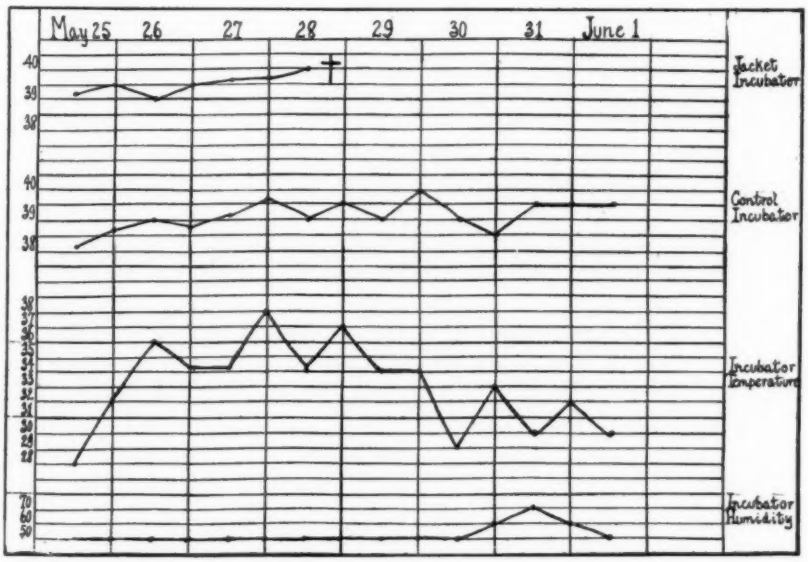

Fig. 2.—Experiment 2.

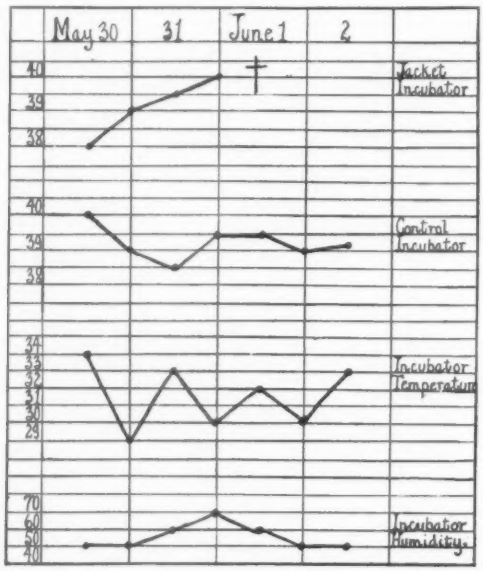

Fig. 3.—Experiment 3.

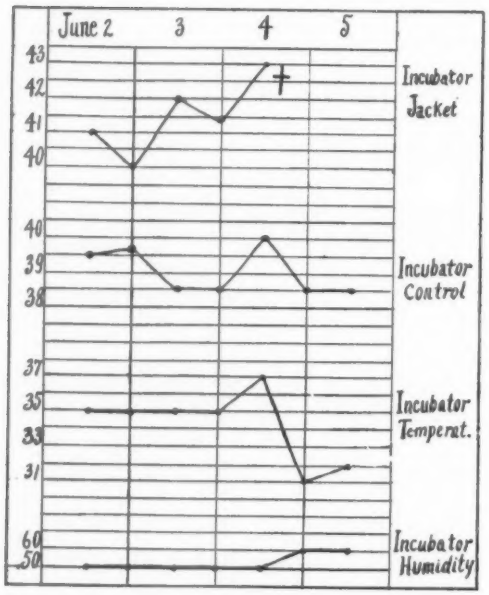

Fig. 4.—Experiment 4.

ature. In a seventh experiment, one without a jacket served as an outside control. Observations were made on the incubator temperature and humidity, and also on the puppies' rectal temperatures. The stools and the general condition of the puppies were also noted. The animals in each experiment received equal amounts of milk as the only food. They were usually fed from the bottle, having been weaned shortly before the experiment began. Water was given freely.

Jackets.—The jacket (except in Experiments 1 and 2, see protocols) was made of two layers of Canton flannel, sewed about the trunk, shoulders and hips of the puppy. The head, upper part of the neck, legs and tail were uncovered. A single layer of adhesive plaster was placed over the flannel to make it fit more snugly. The jackets allowed freedom of movement and respiration.

Incubator.—The incubator was one that had been previously used for premature infants. The temperature in this incubator is maintained by a hot water pan, heated from without by means of a gas burner. The automatic temperature control was not especially accurate. In addition, it was necessary to open the door at periods for feeding, temperature taking and cleaning. No special measures were taken to regulate the humidity nor to insure ideal ventilation. The latter depends on an air inlet of 7 cm. diameter at the bottom of the incubator, and an outlet flue of the same diameter at the top of the chamber.

The accompanying charts show the rectal temperatures of the incubator puppies (jacketed and control), the incubator temperature and humidity; + indicates the death of the animal.

Reference to the charts shows that in each one of the eight experiments the jacketed animal died, hyperthermia being noted in each instance. In Experiments 1, 2, 3, 4, 7 and 8, the jacketed animal died within eighty-four hours. It is seen also that the rectal temperature of the incubator jacketed puppy was usually higher than that of the incubator control animal. In Experiment 5 and Experiment 6 the jacketed animals seemed considerably more resistant, the puppy in Experiment 5 living ten days, during which time two control animals died. In Experiment 6 the animal lived nine days, in which period one control puppy succumbed. In Experiment 5 the first control puppy was unusually fat, the second showed ill effects almost immediately after being put into the incubator and died within twenty-four hours. The first control animal in Experiment 6 showed blood in the stools a few hours before death and at post mortem an intussusception was found about 5 cm. long in the ascending colon. The visceral peritoneum was not adherent but the mucosa was found somewhat necrotic. A dark red area was found about 5 cm. further along in the intestine. It will be seen that the last rectal temperatures before death in the control dogs of Experiment 5 were relatively low compared with those of the other fatal cases.

The control puppies with jackets at room temperature seemed normal. The room temperature was usually about 9° C. lower than that of the incubator. The rectal temperature of these outside control animals was usually about 0.8° C. lower than that of the incubator control animals, and 1.4° C. lower than that of the incubator jacketed puppies.

Stools.—No striking differences were noted in the condition of the stools of the various animals. The stools of the incubator animals were somewhat more frequent and looser than those of the outside control puppies.

Post-Mortem Findings.—Post mortems were made on all puppies which died. (Eight jacketed incubator puppies; three control incubator puppies.)

The principal macroscopic findings were as shown in Table 1.

TABLE 1.—MACROSCOPIC FINDINGS POST MORTEM

Organ	Jacket Incubator Puppies Cases	Control Incubator Puppies Cases
Brain—		
Hyperemia of the meninges	7	2
Hyperemia of the cortex	7	2
Skull not opened	1	1
Thymus—		
Petechial hemorrhages just beneath the surface	6	3
Lungs—		
Hyperemia	6	3
Spleen—		
Soft, deep purple	1	2
Stomach—		
Hemorrhagic areas	2	1
Petechiae	2	..
Duodenum—		
Hyperemia of mucosa	2	..
Petechiae	1	..
Ulcers	1	..
Ileum—		
Petechiae	2	..
Ulcers	..	1
Colon—		
Petechiae	5	2
Intussusception	..	1
Marked subcutaneous fat	..	2

Ascarides were present in the intestines in three cases in which petechiae were found, and were also present in two cases in which no petechiae were found.

Experiment.—A four-months-old puppy without a jacket was kept in the incubator for one month at a mean temperature of 35.9° C. and a mean humidity of 72.5 per cent. A control puppy of the same litter was kept in the room, the mean temperature of which was about 25° C. The animal in the incubator thrived, increasing in weight from 2,020 gm. to 3,390 gm., while the control puppy increased from 1,920 gm. to 2,520 gm. The rectal temperature of the incubator animal was about 1 degree Centigrade higher than that of the control. The latter developed a diarrhea, which doubtless influenced his weight curve.

Experiment.—In the latter part of Experiment 6 a shaved puppy without a jacket was placed in the incubator, in addition to the usual jacketed and unjacketed animals. This shaved puppy seemed more comfortable than the others and his rectal temperature was usually about 0.4° C. lower than that of the unshaved, unjacketed incubator-control puppy.

DISCUSSION

The main means of heat loss in puppies under ordinary conditions are:

1. Radiation and conduction.
2. Evaporation of water.
   (a) From the lungs and tongue.
   (b) From the sweat glands of the paws.
   (c) Through so-called insensible perspiration through the skin of the entire body.

At relatively low temperatures, conduction and radiation play the more important rôle with regard to heat loss. At high temperatures,

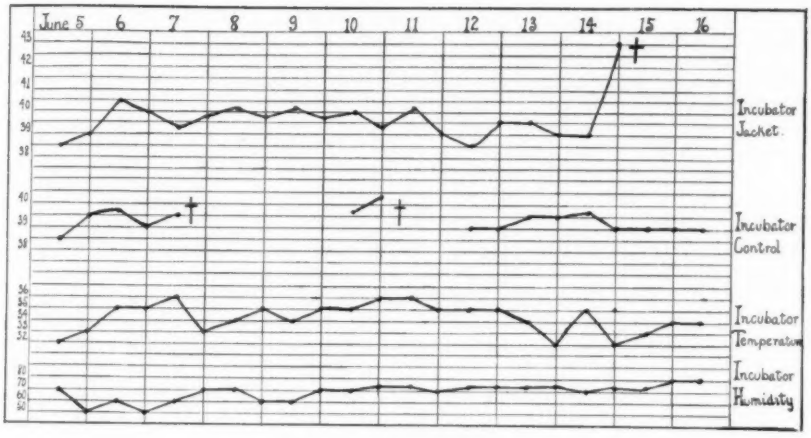

Fig. 5.—Experiment 5.

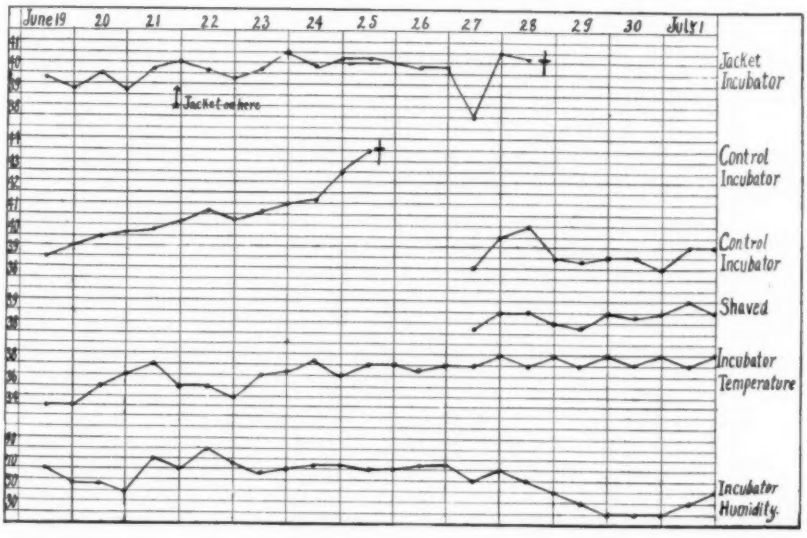

Fig. 6.—Experiment 6.

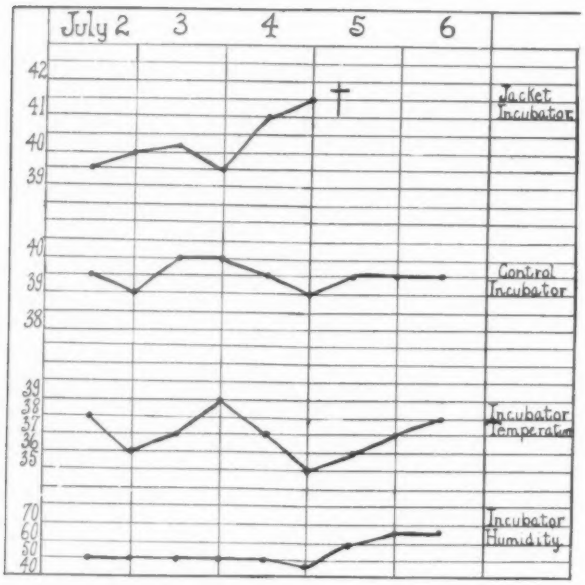

Fig. 7.—Experiment 7.

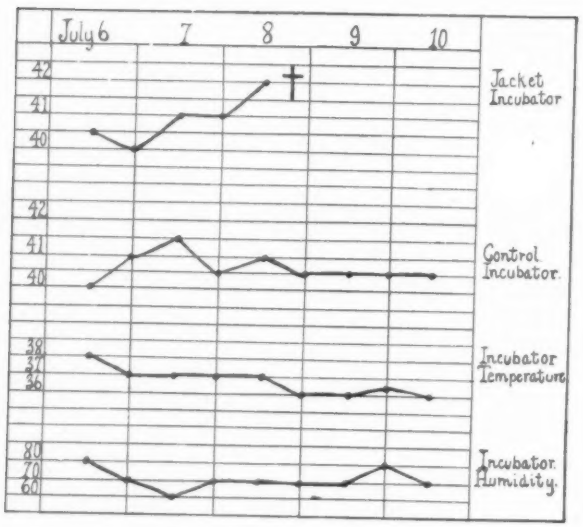

Fig. 8.—Experiment 8.

however, evaporation of water becomes more important. Since puppies perspire only on the paws (Luschsinger[10]), except under very unusual conditions,[11] panting becomes very important at high temperatures in increasing the heat loss by evaporation of water from the lungs and tongue. In our experiments, the jackets interfere only with heat loss through conduction and radiation, and the evaporation of the so-called insensible perspiration. In spite of this, i. e., without influencing appreciably the mechanism of heat loss, most important at high temperatures (evaporation), the interference with the less important factors at high temperatures (radiation and conduction) led to deleterious results.

Clothing interferes with heat loss by conduction, radiation and possibly evaporation of insensible perspiration in the infant as well as in the puppy. While conduction and radiation become less important as a means of heat loss, as the surrounding temperature approaches that of the body, one may deduct by analogy (cf. our experiments) that this relatively slight inhibition of heat loss may suffice to induce untoward results. In addition some infants, at least, perspire from the sweat glands of the body in general at high temperatures, and the clothing in such cases, if not freely permeable, may also interfere with heat loss by evaporation.

A portion of the infant deaths, especially those occurring in the first hot days of summer, is probably due to heat stasis per se (Finkelstein,[12] Rietschel,[6] Liefmann and Lindemann,[8] L. F. Meyer,[13] etc.). It seems probable to us that there are many more cases in which disturbed heat regulation, though not sufficient to produce death, suffices to cause disturbances in the body functions, especially those of the digestive tract. The following case, reported by Wolff,[14] seems to support this view:

A 2-months-old infant prematurely born (seventh month), came into the hospital suffering with a diarrhea. The infant was put into an incubator, the food regulated and the condition soon cleared up. Later, the child was removed to a crib and surrounded by hot-water bottles. One day, by accident, the temperature of the water in the bottles was too high and within a few hours the child developed a diarrhea with frequent green liquid defecations and vomiting. The fontanel became sunken, the child became semicomatose, the temperature rose to 42 C., and the clinical picture was that of Finkelstein's alimentary

10. Luschsinger: Cf. Hermann's Handbuch der Physiologie, Leipzig, 1883, p. 427.

11. Goltz and Ewald (Pflüger's Arch. f. d. ges. Physiol., 1896, lxiii, 370), say that after the dog's cervical cord had been cut and the animal was held in the incubator, there soon became noticeable a layer of perspiration over the entire haired surface of the body excepting the head. The animal was not overheated, for there was neither tachypnea nor tachycardia.

12. Finkelstein: Ueber den Sommergipfel der Säuglingssterblichkeit, Deutsch. med. Wchnschr., 1909, xxxii.

13. Meyer, L. F.: Die Morbidität und Mortalität der Säuglinge im Sommer, 1911. Deutsch. Naturforsch u. Aertzte, 1911, Wiesbaden, 1912, xxvii, 55.

14. Wolff: Beitrag zur Frage der Sommersterblichkeit der Säuglinge, Jahrb. f. Kinderh., 1913, lxxvii, 569.

intoxication. On removal of the external heat and after giving a bath, but without any other change whatsoever, the infant's condition returned to normal in a very short time.

In a majority of cases of intestinal disturbance caused by a disturbed heat equilibrium, therapeutic results cannot be expected of measures intended to increase heat loss. The damage done to the infant organism is already too great. Results are, however, to be expected from prophylactic measures. Rubner[1] has shown that in adults, with food, health and clothing at an optimum, comfort is experienced with the surrounding temperature of 16 to 25 C. In the adult, clothing is almost automatically adjusted to the feeling of comfort; whereas in the infant, where the question of clothing is even more important because of the relatively much greater surface area, this regulation must depend on the judgment or whim of the caretaker. Sleeplessness, restlessness, and increased irritability are signs of discomfort in the infant that is overclothed. We are gathering statistics, to be published later, which thus far show surprisingly little difference between the weights of indoor clothing commonly worn by infants during the summer and winter.

Infected food as an element in summer mortality of infants, once considered the prime factor, has within the past few years been given a secondary place by many investigators. This change in view has probably come about principally because of the fact that where good milk has been furnished as the only food, less attention being paid to the other factors, the death rate has been disappointingly little influenced (Thiemich,[15] Keller,[16] Liefmann,[17] Rietschel,[18] etc.).

SUGGESTIONS

In considering what steps may be taken to prevent or reduce the high summer death rate of infants, we believe that a number of factors must be adjusted:

1. Decrease heat production by giving less food, more suitable food and more water.
2. Increase heat elimination by less and more permeable clothing, better circulation of air and frequent baths.
3. Guard against infection (through food and otherwise).

We wish to express our thanks to Mrs. Henry F. Helmholz for her kind assistance in preparing the charts.

15. Thiemich: Die Sozialen Beziehungen und Aufgaben der Kinderheilkunde, Deutsch. med. Wchnschr., 1913, xxxix, 1739.
16. Keller, A.: Der Stand der Säuglingsfürsorge. Stenogr. Ber. u. d. Verhandl. d. Deutsch. Kong. f. Säuglingsschutz, Berlin, 1907, p. 27.
17. Liefmann: Die Bedeutung sozialer Momente für die Säuglingssterblichkeit, Ztschr. f. Hyg., 1909, lxii, 199.
18. Rietschel: Sommerhitze, Wohnungstemperatur und Säuglingssterblichkeit, Ztschr. f. Kinderh., 1911, i, 546; Zur Aetiologie des Sommer brechdurchfalls der Säuglinge, Monatschr. f. Kinderh., 1911, ix, 39.

PROTOCOLS

TABLE 2.—DATA OBTAINED IN EXPERIMENT 1

Date	Hour	Incubator Puppies		Outside Control Puppy Temp. °C.	Incubator Temperature	Incubator Humidity Per Cent.	Room Temp.
		Temp. °C. Jacketed	Temp. °C. Control				
May 20	4 p. m.	39.5	39.5	38.2	37	50	28.3
May 21	8 a. m.	40.	38.5	38.6	34	70	
	4 p. m.	40.5	39.5	39.6	35	70	24.4
May 22	8 a. m.	....*	38.5	38.8	35	70	
	4 p. m.	....	39.	39.4	35	70	31.1

* Found dead.

The outside control animal had no jacket on in Experiment 1. The jacket of the incubator puppy was wrapped with a thin layer of plaster-of-Paris gauze (instead of adhesive plaster as in Experiments 2, 4, 5, 6, 7, 8). The weight of jacket = 90 gm. Stools of all puppies practically normal.

Post mortem on jacketed incubator puppy:

Skull: Meninges somewhat injected, cortical vessels prominent.

Abdominal Cavity: Peritoneal surfaces smooth, moist, shiny.

Pleural Cavity: Surfaces smooth, moist, shiny.

Pericardial Cavity: Normal.

Heart: No thrombi, no hemorrhages.

Lungs: Marked hyperemia, no consolidation, crepitation throughout.

Thymus: Pale pink, eight to ten pinhead sized hemorrhages just beneath the surface.

Spleen, liver, pancreas, kidneys and bladder appear normal.

Stomach: Distended with gas, contains coagulated milk, mucosa pale.

Intestines: Somewhat distended, mucosa of small intestines pale pink.

Colon and Rectum: Somewhat hyperemic. There are from forty to fifty pinhead-sized hemorrhages in the mucosa and a few streaks of free blood on the mucosa of the rectum. Eight ascarides in the small intestine.

TABLE 3.—DATA OBTAINED IN EXPERIMENT 2

Date	Hour	Incubator Puppies		Outside Control Puppy (Jacketed) Temp. °C.	Incubator Temperature ° C.	Incubator Humidity Per Cent.	Room Temp.
		Temp. °C. Jacketed	Temp °C. Control				
May 25	4 p. m.	39.2	38.2	38.2	29	50	23.3
May 26	8 a. m.	39.5	38.8	39.0	28	50	23.9
	4 p. m.	39.0	39.0	39.2	32	50	23.3
May 27	8 a. m.	39.5	38.8	39.0	36	50	23.3
	4 p. m.	39.8	39.2	38.4	34	50	23.3
May 28	8 a. m.	39.8	39.7	38.4	34	50	27.2
	4 p. m.	40.0	38.8	38.2	37	50	27.2
May 29	8 a. m.	....*	39.5	39.1	34	50	30.5
	4 p. m.	....	39.0	39.0	37	50	28.9

* Found dead.

Jackets in Experiment 2 were made of a layer of cotton between gauze and covered with a layer of flannel. Weight of jacket = 70 gm. Stools were slightly loose; the outside control had mucus in the stools and once there was a tinge of blood.

Post mortem on the jacketed incubator puppy:

Skull: Meninges injected; cortical vessels prominent.

Abdominal, pleural and pericardial cavity surfaces normal.

Heart: Normal (no thrombi, no hemorrhages).

Lungs: Marked hyperemia, no consolidation, excess of blood-stained fluid expressed on incision.

Thymus: Pale pink, a number of pinhead-sized hemorrhages just below surface in center of lobules.

Spleen, liver, pancreas, kidneys, bladder and adrenals appear normal.

Stomach: Two hemorrhagic areas the size of a dime. No ulcers.

Duodenum: Mucosa pink, no hemorrhages.

Jejunum and ileum normal.

Colon and Rectum: A few small hemorrhagic areas; streaks of blood on the mucosa. No ulcers seen.

TABLE 4.—DATA OBTAINED IN EXPERIMENT 3

Date	Hour	Incubator Puppies		Outside Control Puppy Temp. °C.	Incubator Temp. ° C.	Incubator Humidity Per Cent.	Room Temp. ° C.
		Temp. °C. Jacketed	Temp. °C. Control				
May 30	4 p. m.	38.0	40.0	39.0	34	50	21.7
May 31	8 a. m.	39.0	39.0	38.6	29	50	24.4
	4 p. m.	39.5	38.5	38.4	33	60	23.9
June 1	8 a. m.	40.0	39.5	39.2	30	70	24.4
	4 p. m.	....*	39.5	39.4	32	60	25.5

* Found dead.

The jackets consisted of two layers of canton flannel and one layer of adhesive plaster. Jacket of the incubator animal = 60 gm. The animal was found dead under the incubator basket.

Postmortem on incubator (jacketed) puppy:

Skull: Meninges and cortical vessels slightly injected.

Abdominal, pleural and pericardial cavity surfaces normal.

Heart: Normal.

Lungs: Marked hyperemia, somewhat firm in some parts, but no pneumonic areas.

Thymus: Small petechial hemorrhages just beneath the surface in the center of the lobules.

Spleen, liver, pancreas and bladder appear normal.

Stomach: Normal. Small Intestine: Normal. Colon and Rectum: A number of small hemorrhages in the mucosa. No ascarides.

TABLE 5.—DATA IN EXPERIMENT 4

Date	Hour	Incubator Puppies		Outside Control Puppy Temp. °C.	Incubator Temp. ° C.	Incubator Humidity Per Cent.	Room Temp. ° C.
		Temp. °C. Jacketed	Temp. °C. Control				
June 2	4 p. m.	41.0	39.5	39.4	35	50	23.3
June 3	8 a. m.	40.0	39.5	39.2	35	50	23.3
	4 p. m.	42.0	38.5	39.0	35	50	25.5
June 4	8 a. m.	41.5	38.5	38.6	35	50	23.3
	4 p. m.	43.0	40.0	39.4	37	50	27.8
June 5	8 a. m.	....*	38.5	38.2	31	60	27.8
	4 p. m.	....	38.5	38.0	32	60	28.9

* Found dead.

The jackets were as described in Experiment 3. The incubator animals developed a diarrhea; the last few stools of the jacketed puppy contained mucus and blood.

Post mortem on jacketed incubator puppy:

Skull: Meninges and cortical vessels slightly injected.

Abdominal, pleural and pericardial cavity surfaces normal.

Heart: Normal.

Spleen, pancreas, liver, kidneys and bladder appear normal.

Stomach: Mucosa pale.

Duodenum: Mucosa hyperemic.

Jejunum and Ileum: Normal. Colon and Rectum: Small hemorrhages in mucosa. No ascarides.

TABLE 6.—DATA OBTAINED IN EXPERIMENT 5

Date	Hour	Incubator Puppies		Outside Control Puppy Temp. °C.	Incubator Temp. ° C.	Incubator Humidity Per Cent.	Room Temp. ° C.
		Temp. °C. Jacketed	Temp. °C. Control				
June 6	8 a. m.	39.0	39.5	38.5	33	50	23.9
	4 p. m.	40.5	39.8	38.2	35	60	23.9
June 7	8 a. m.	40.0	39.0	38.4	35	50	23.3
	4 p. m.	39.2	39.5	38.6	36	60	23.3
June 8	8 a. m.	39.8	....*	38.2	33	70	24.4
	4 p. m.	40.2	....	38.4	34	70	23.3
June 9	8 a. m.	39.8	....	39.0	35	60	23.3
	4 p. m.	40.4	....	39.2	34	60	23.3
June.10	8 a. m.	39.6	39.8	39.2	35	70	21.1
	4 p. m.	40.0	40.4	39.4	35	70	22.2
June 11	8 a. m.	39.2	....*	38.2	36	72	23.3
	4 p. m.	40.2	....	39.0	36	72	21.1
June 12	8 a. m.	39.0	....	....	35	70	
	4 p. m.	38.5	39.0	....	35	75	23.9
June 13	8 a. m.	39.5	39.0	....	35	75	24.4
	4 p. m.	39.5	39.5	....	34	75	23.3
June 14	8 a. m.	39.0	39.5	....	32	75	23.3
	4 p. m.	39.0	39.8	....	35	70	23.3
June 15	8 a. m.	43.0	39.0	....	32	75	
	4 p. m.	....*	39.0	....	33	75	23.9
June 16	8 a. m.	....	39.0	....	34	80	
	4 p. m.	....	39.0	....	34	80	23.9

* Found dead.

The jackets were as in Experiment 3. The stools of the incubator animals were, at times, thin and yellow, with mucus occasionally. The first control animal in the incubator was exceptionally fat, and the second incubator control animal appeared sick a few hours after being put into the incubator.

Necropsy on the jacketed incubator puppy:

Skull: Meninges and cortical vesseis hyperemic.

Abdominal, pleural and pericardial cavity surfaces normal.

Heart: Normal.

Lungs: Nearly collapsed, congestion and bogginess of the lower lobes, bronchi normal.

Thymus: Exceptionally small. No hemorrhages.

Spleen, pancreas, liver, kidneys and bladder appear normal, except for a slight hyperemia of the papillae of the kidneys.

Stomach: Mucosa hemorrhagic in pyloric region.

Duodenum, jejunum and ileum slight hyperemia of mucosa.

Colon and Rectum: More marked hyperemia.

Necropsy on first incubator control puppy of Experiment 5:

Skull: Meningeal and cortical vessels injected.

Abdomen: Much distended with gas.

Subcutaneous Tissues: Emphysematous; marked adiposity.

Abdominal, pleural and pericardial cavity surfaces normal.

Peritoneal Cavity: The contents of the stomach and three ascarides were found in the peritoneal cavity (probably post mortem).

Thymus: Many hemorrhagic spots beneath the surface, in the center of the lobules.

Heart: Anemic; otherwise normal.

Lungs: Hyperemic.

Spleen, pancreas, kidneys and bladder normal. Liver soft; some post mortem changes.

Stomach: There is a 5 cm. tear in the fundus region, from which the gastric contents escaped into the peritoneal cavity.

Intestines: Show post mortem changes and are very much distended with gas.

Post mortem on second incubator control puppy:

Skull: Not opened.

Peritoneal, pleural and pericardial cavity surfaces normal except for a slight congestion of the pleural surfaces.

Heart: In systole; a few petechial hemorrhages under the epicardium; myocardium is pale.

Thymus: Numerous hemorrhagic areas (flaxseed size) under the surface in the center of lobules.

Spleen and liver congested and friable. Kidneys pale. Bladder and adrenals are normal.

Stomach: The mucosa is pale.

Small Intestine: Mucosa is pale.

Colon: Mucosa is pale.

Rectum: Many small hemorrhages in the mucosa.

Five ascarides in the ileum and colon.

TABLE 7.—Data Obtained in Experiment 6

Date	Hour	Incubator Puppies		Outside Control (Jacketed) Temp.°C.	Incubator Temp. ° C.	Incubator Humidity Per Cent.	Room Temp. ° C.
		Temp. ° C.	Temp. ° C.				
June 19	4 p. m.	39.8	39.0	38.8	34	70	23.3
June 20	8 a. m.	39.3	39.5	38.6	34	58	23.3
	4 p. m.	40.0	40.0	38.4	35	58	23.3
June 21	8 a. m.	39.3	40.3	38.4	36	50	23.3
	4 p. m.	40.2 Jacket on here	40.3 Control	38.6	37	80	24.4
June 22	8 a. m.	40.5	40.7	38.6	35	70	23.9
	4 p. m.	40.3	41.1	38.8	35	90	26.1
June 23	8 a. m.	39.8	40.8	38.4	34	78	25.0
	4 p. m.	40.3	41.3	38.9	36	70	26.7
June 24	8 a. m.	41.0	41.5	38.6	36.5	70	26.7
	4 p. m.	40.3	41.7	38.6	37.6	77	24.3
June 25	8 a. m.	40.7	43.0	38.8	36	77	23.3
	4 p. m.	40.7	44.0	38.2	37	70	23.3
June 26	8 a. m.	40.5	....*	38.4	37	70	23.3
	4 p. m.	40.3	....	38.6	36.6	75	23.3

TABLE 7.—(*Continued*)

Date	Hour	Incubator Puppies		Outside Control (Jacketed) Temp. °C.	Incubator Temp. ° C.	Incubator Humidity Per Cent.	Room Temp. ° C.
		Temp. ° C.	Temp. ° C.				
			New control	Shaved incubator puppy			
June 27	4 p. m.	38.0	38.5	38.3	37	70	
June 28	8 a. m.	41.0	40.0	39.0	37	55	
	4 p. m.	40.7	40.5	39.0	38	40	
June 29	8 a. m.	....*	39.0	38.5	37	38	
	4 p. m.	....	38.8	38.4	38	30	
June 30	8 a. m.	....	39.0	39.0	37	30	
	4 p. m.	....	39.0	38.8	38	40	
July 1	8 a. m.	....	38.5	39.0	37	50	

* Found dead.

In Experiment 6 neither incubator puppy had a jacket for the first three days; on the fourth day the usual jacket was put on one of the animals. The control died seven days after being put into the incubator. Blood was found in this animal's stools a few hours before death (intussusception; see notes below). The jacketed incubator animal died two days later. In the latter part of this experiment three animals were in the incubator: one shaved, one with a jacket and the normal control. The shaved animal showed less distress than the other two. The first incubator control puppy and the jacketed incubator puppy had watery stools part of the time; the second control puppy and the shaved puppy had normal stools.

Post mortem on jacketed incubator puppy of Experiment 6:

Skull: Meningeal and cortical vessels injected.

Peritoneal, pleural and pericardial cavity surfaces normal.

Heart: Normal.

Lungs: Mottled purple areas, the rest of the lungs somewhat hyperemic.

Thymus: Petechial hemorrhages beneath the surface in the center of the lobules.

Spleen, pancreas, liver and bladder normal.

Kidneys: Hyperemia of the papillae.

Stomach: A few hemorrhagic spots near the pylorus.

Duodenum: A few small ulcers, one about the size of a flaxseed.

Jejunum: Normal. Ileum: There are a few petechiae in the lower part.

Colon: There are about twenty petechiae in the cecal region; no macroscopic ulcers.

Post mortem on first control incubator puppy of Experiment 6:

Marked adiposity.

Skull: Meninges and cortical vessels are injected.

Peritoneal, pleural and pericardial cavity surfaces normal.

Heart: Normal.

Lungs: Congestion of the lower lobes; no consolidation.

Thymus: There are about twelve petechiae just beneath the surface. Spleen congested.

Pancreas, liver, kidneys and bladder normal.

Stomach: A few petechiae in the mucosa.

Ileum: A few pinhead-sized ulcers. Colon: An intussusception, about 5 cm. long involves the cecum and ascending colon. The lumen is patent for a small sound. The intussusception is easily reduced; the mucosa there is somewhat necrotic. The visceral peritoneum is smooth and shiny. An edematous dark red area is found about 5 cm. from the intussusception. The appendix is edematous with a marked injection of its mucosa.

TABLE 8.—Data Obtained in Experiment 7

Date	Hour	Incubator Puppies		Outside Control Puppy Temp. °C.	Incubator Temp. °C.	Incubator Humidity Per Cent.
		Temp. °C. Jacketed	Temp. °C. Control			
July 2	8 a. m.	....	39.5	39.0	38	50
	4 p. m.	39.5	39.5	38.8	38	50
July 3	8 a. m.	40.0	39.0	38.5	36	52
	4 p. m.	40.3	40.0	39.5	37	50
July 4	8 a. m.	39.5	40.0	39.0	39	50
	4 p. m.	41.0	39.5	40.0	37	47
July 5	8 a. m.	42.0	39.0	39.8	35	60
	4 p. m.	....*	39.8	38.5	36	70
July 6	8 a. m.	....	39.8	39.5	37	68

* Found dead.

The stools of the jacketed incubator puppy were loose at times, with, occasionally, particles of mucus. The stools of the control puppies were normal. Jackets as in Experiment 3.

Post mortem on jacketed incubator puppy of Experiment 7:

Skull: Not opened.

Peritoneal, pleural and pericardial cavity surfaces normal.

Heart: There are three lintel sized hemorrhages beneath the epicardium.

Lungs: There is some congestion of the lower lobes.

Thymus: A number of small petechiae just beneath the surface.

Spleen, liver, pancreas, kidneys, adrenals and bladder normal.

Small Intestine: Excessive mucus with edema of the mucosa. Three ascarides in the small intestine.

Colon: Serosa hyperemic; mucosa normal.

TABLE 9.—Data Obtained in Experiment 8

Date	Hour	Incubator Puppies		Incubator Temp. °C.	Incubator Humidity Per Cent.
		Temp. °C. Jacketed	Temp. °C. Control		
July 6	4 p. m.	40.5	40.0	38	80
July 7	8 a. m.	40.0	41.0	37	70
	4 p. m.	41.0	41.5	37	60
July 8	8 a. m.	41.0	40.5	37	70
	4 p. m.	42.0	41.0	37	75
July 9	8 a. m.	....*	40.5	36	75
	4 p. m.	....	40.5	36	75
July 10	8 a. m.	....	40.5	36.5	80
	4 p. m.	....	40.5	36	70

* Found dead.

Jacket as in Experiment 3.

Post mortem on jacketed incubator puppy of Experiment 8:

Skull: Meningeal and cortical vessels of the left hemisphere injected; those of the right anemic, with a marked edema of the brain.

Peritoneal, pleural and pericardial cavity surfaces normal.

Heart: Small and anemic.

Lungs: Pale, crepitate throughout; lintel-sized deep red areas in the dependent portions.

Thymus: Five small petechiae just beneath the surface.

Spleen: Is hyperemic and soft.

Liver, pancreas, kidneys, adrenals and bladder normal.

Stomach: Contains bile-stained fluid; there are about 40-50 petechiae in the mucosa near the fundus.

Small Intestine: Three flaxseed-sized hemorrhages in the mucosa, no ulcers. Three ascarides in the small intestine.

Colon: Four small hemorrhagic areas in the lower part of the colon.

TUBERCULOUS TUMORS OF THE BRAIN

REPORT OF A CASE AND BRIEF SUMMARY OF THE LITERATURE*

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Tuberculous tumors are the most frequent of all cerebral tumors. During childhood they are especially common, forming over 50 per cent. of all brain tumors.[48] In 300 cases of brain tumor in persons under 18 years of age, reported by Starr,[45] 152 were tuberculous. Peterson[37] reports 335 cases of which 166 were tuberculous.

Brain tubercle is almost always secondary to some other tuberculous lesion. In about 25 per cent. of the cases it is multiple.[48]

Tubercles are most commonly found in the cerebellum. The pons is also a rather frequent seat. Larger tubercles are comparatively infrequently found in the cortex; when found they are usually situated in the paracentral lobe.[48] The situation in Starr's[45] 152 cases was as follows: cerebellum 47, pons 19, corpora quadrigemina and crura 16, basal ganglia and lateral ventricles 14, cortex cerebri 13, centrum ovale 6, medulla 2.

The tubercle usually forms about a blood vessel, caseation occurring in the center. It is sharply defined from the surrounding brain substance, which undergoes softening.

The following case has many points of interest, and is therefore reported.

J. S., a colored boy of not quite 7, had been an inmate of the St. Elizabeth's Home for three years. Nothing was known of his history previous to his entrance into the institution. When first seen in June, 1912, the child was fairly well nourished, but pale and undersized. His intelligence was as good as that of the average child of his age in the institution. While in the home the child had not complained and had had no illness. The child had a convulsion in June, 1912, and it was said, passed a roundworm. A blood examination showed a marked eosinophilia. Santonin and calomel were given; he did not, however, pass any worms. Convulsions epileptiform in character occurred at rather irregular intervals. These convulsions always set in with a spasm of the right arm and leg. Just before the occurrence of a convulsion, the child would run up to the Sister and say, "Sister, I'm sick." A blood examination made some-

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what later did not show an eosinophilia. The Wassermann reaction was positive. The child showed general glandular enlargement, but nothing important was found on physical examination at this time. The patient was given active antisyphilitic treatment over a period of five months without any improvement. During the summer and autumn of 1912 the convulsions became more and more frequent, the child's appetite failed; he lost weight rapidly, became markedly emaciated and very weak. Toward the end of autumn the convulsions became very violent in character, always coming on with twitchings of right arm and leg. On Jan. 11, 1913, following a violent convulsion, the patient became acutely ill, the temperature rose, the respiration became rapid and labored. On physical examination, patches of crepitation were found over the whole right lung. Bronchial breathing was also noticed at various points. These pneumonic symptoms continued; the patient gradually became weaker and weaker. On the 21st the abdomen became rigid, distended, tender and dulness in the flanks indicated fluid. The child died Jan. 24, 1913, his illness having covered a period of from seven to eight months.

Jan. 24, 1913, at necropsy, the body was that of a markedly emaciated boy of about 7 years. The right lung was involved in an extensive caseous pneumonia and the pleurae were thickened and adherent over most of their surface. On section small and medium sized cavities could be seen in the upper lobe. The left pleurae were thickened and adherent over most of the lung, especially so near the apex. The left lung itself, with the exception of the apex, was free from tuberculosis. The bronchial lymph nodes were everywhere enlarged, some to the size of a walnut, quite dense and on section showed caseous centers. Microscopically, immense fibroid tubercles, some of which were hyaline and showed slightly caseous centers, were found in the left apex; they appeared to be inactive and to denote a very chronic course.

The heart showed no abnormalities.

The abdominal cavity contained a large quantity of yellowish turbid fluid and there were extensive peritoneal adhesions present binding coils of intestines to each other. Miliary tubercles were in evidence on the visceral peritoneum. The mesenteric nodes, both grossly and microscopically, showed extensive caseation.

The liver was large, and had the typical yellowish, waxy hue of a fatty liver. To the naked eye there was no evidence of tuberculosis, but microscopically small tuberculous foci with beginning caseation were found, mainly in the interlobular connective tissue but also intralobularly. The capsule showed a chronic inflammatory thickening with hyaline changes.

The spleen was slightly enlarged and congested, and near the hilum a small nodule about the size of a pea was found, which proved to be an accessory splenule. The capsule was thickened, and even to the naked eye showed fine pin head nodules of miliary tubercles. Hyaline changes in the trabeculae and in the blood vessel walls were found microscopically. The accessory splenule showed a few caseous and cellular foci and hyaline changes.

The kidneys on microscopical examination showed marked cloudy swelling and also fatty degeneration of the epithelium of the convoluted tubules and slight proliferative changes in some of the glomeruli, hyaline changes in the blood vessel walls, and inflammatory thickening of the capsule.

The pancreas showed no lesion of interest.

On running the fingers over the convexity of the brain, a number of hard shotlike elevations varying in size from a pea to a medium sized marble were felt in different portions of both cerebral hemispheres. The pia mater was everywhere congested, and over the areas just described appeared hazy and opaque, and was adherent. With these exceptions, the meninges were free from disease. On removal of the meninges and on closer examination, these areas were found to consist of dense white or grayish masses imbedded in the cerebral hemispheres. They apparently had little connection with the surrounding brain tissue, for they could be shelled out very easily.

Their exact location and size are indicated on the diagrams. The left cerebral hemisphere contained a number of conglomerate foci, forming a large mass situated in the ascending parietal and partly in the superior marginal and superior parietal convolutions opposite the leg, trunk and arm motor areas. As shown in the drawing (Fig. 1 *a*) the mass appeared on the surface and involved the gray matter in its upper part only, while the white matter beneath was involved to a greater extent, impinging on the gyri within the rolandic fissure.

The right cerebral hemisphere contained four masses of various dimensions, three of which were apparent on the external surface of

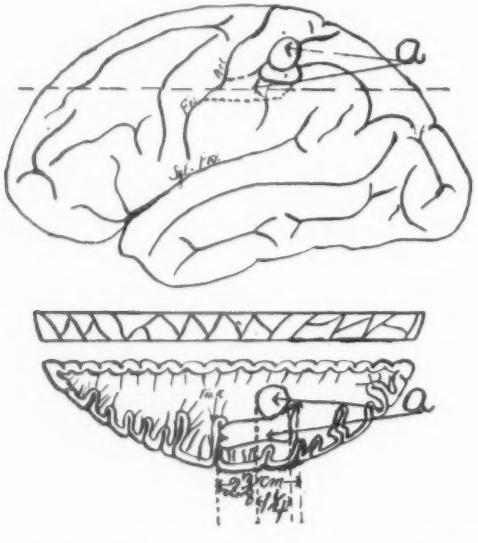

Fig. 1.—Left cerebral hemisphere: *a*, fused mass, maximum depth 2⅞ cm., width 2¾ cm., length 2⅜ cm.; dotted lines in upper drawing indicate where tumor passes beneath gray matter at about an angle of 10 degrees both anteriorly and externally. The diagrams in this and Figure 2 were drawn directly from the specimen, after the method employed by draftsmen in drawing a plan of an object. The disconnected horizontal lines passing across the cerebral hemisphere indicate the planes of section, which below are projected, the horizontal surfaces thus exposed, and at times the perpendicular edges of the corresponding sections.

the brain; the fourth, the largest, appeared only on section. As seen in the drawing (Fig. 2 *a*) one of these occupied the upper part of the midfrontal convolution and was well forward; another (Fig. 2 *b*) the lower part of the external surface of the occipital lobe (mid-occipital convolution); and a third (Fig. 2 *c*) was situated about the parieto-occipital sulcus, both on the external and mesial surfaces. The fourth mass (Fig. 2 *d*) was of large dimensions, conical in shape, its base involving the temporal operculum and extending across the limiting sulcus, involved also the upper part of the insula, the claustrum and

external capsule. Its tapering extremity passed upward lying posterior to the fissure of Rolando and between it and the sylvian fissure.

These masses could easily be seen to be made up of a number of conglomerate foci, fused together. On the whole, they were dense yellowish-white or grayish in color, and generally showed some caseation within the center. Histologically they showed the ordinary changes produced by the tubercle bacillus. An extensive caseous center

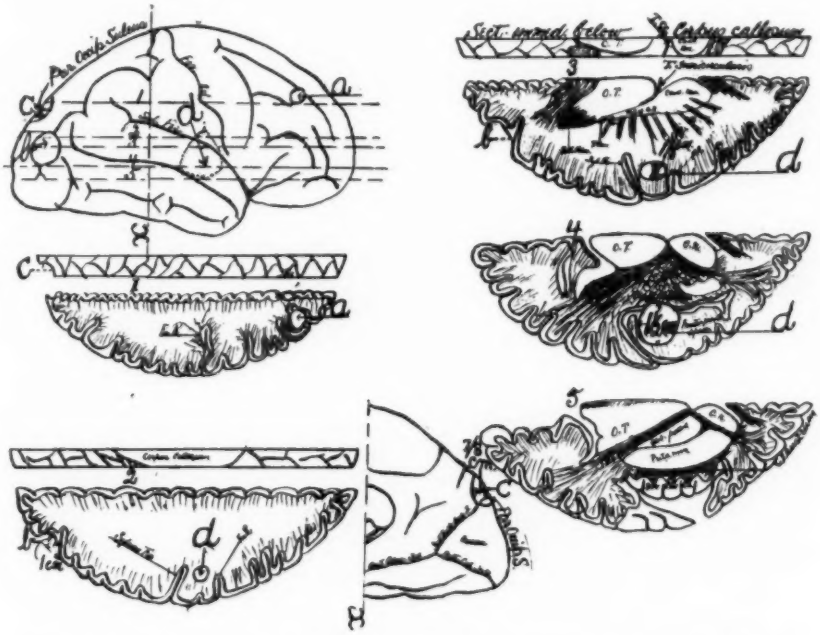

Fig. 2.—Right cerebral hemisphere: *a*, mass, diameter ¾ cm., depth 1 cm.; *b*, mass, diameter 1¼ cm., depth 1 cm.; *c*, mass, diameter 1⅜ cm., depth ½ cm.; *d*, mass (appears in dotted lines in lateral view of cerebral hemisphere), from base to apex 3¼ cm., diameter of base 1½ cm. In the fourth cross sectional view, the base of this mass is shown occupying the fronto-parietal operculum crossing the limiting sulcus of Reil, and invading the insula. The claustrum cannot be made out here with the naked eye. The fifth cross sectional view is below the level of the mass (*d*) and shows the normal relation of the parts.

The central figure below represents the mesial surface of Figure 2 posterior to line x.

was generally present, surrounded by a more cellular periphery and in some instances by a small margin of brain substance, which showed reactive inflammation.

The presence of numerous medium sized blood vessels in various stages of the pathological process was noticed. In the center of the nodules, the vessel-walls were extensively involved in the caseous process and contained necrotic and caseous thrombi. At the periphery

in the areas less involved the vessel-walls showed periarteritis and endarteritis and hyaline changes, and most of them also contained necrotic thrombi. Immediately surrounding the necrotic zone, epithelioid cells, partly degenerated, were found in large numbers. These in turn were surrounded by an area of reactive inflammation presenting perivascular round cell infiltration, with numerous plasma cells, round cells, variously shaped endothelial cells, and marked vascular congestion. The tuberculous giant cell with one or two exceptions was not found. The pia mater was caseous directly over the nodules, while immediately surrounding these areas, it showed merely a mild reactive inflammation. The rest of the pia mater over the brain was practically free from disease. Sections stained for tubercle bacilli showed them in great abundance. Innumerable tubercle bacilli abounded in and about the walls of several medium sized blood vessels. The arterial walls themselves had undergone hyaline degeneration, and endarteritis and periarteritis were present. The bacilli were so numerous and occurred in such large clumps that they could easily be recognized (Fig. 3 as red masses in the walls and for some distance about the blood vessels. With the oil-immersion lens, the individual bacilli could easily be seen making up these clumps; in a number of places they were found, within the lumen of the blood vessel, among the red blood cells (Fig. 4). They were also found with ease, scattered about caseous and inflammatory areas, occurring singly or in clumps, but in much less abundance than about the blood vessels.

The presence of immense numbers of tubercle bacilli in and about a large blood vessel of a caseous bronchial lymph node was noted, although in sections of the lung and other tuberculous organs tubercle bacilli were found singly and with great difficulty.

In view of the double plus Wassermann, a careful search for the *Spirochacta pallida* was made in the various diseased organs, but with negative results.

Although careful search was made of the large blood vessels at the base of the brain both grossly and microscopically, no evidence of tuberculosis was found. Cultures from lung grown on Löfflers serum medium showed the *Streptococcus mucosus*. The streptococci appeared in diplococcus form surrounded by a large conspicuous capsule.

In the case reported by us, the original focus from which the infection spread was possibly a latent focus in the fibroid apex of the left lung or a bronchial lymph node. The infection was early carried to the brain. The twitchings of the right arm and leg are easily explained by the tumor in the left ascending parietal convolution.

A tuberculous pneumonia and a general miliary tuberculosis occurred as a terminal condition, as is shown by the pathological find-

ings and the tubercle bacilli in the blood. This is the usual termination, as seen in most of the cases reported in the literature. The case is a typical one of solitary tubercle, or tuberculoma of the brain. All sections of tissues for microscopic examination were fixed in 10 per cent. of the commercial formaldehyd and embedded in celloidin. The staining process employed to demonstrate the tubercle bacillus in the tissues

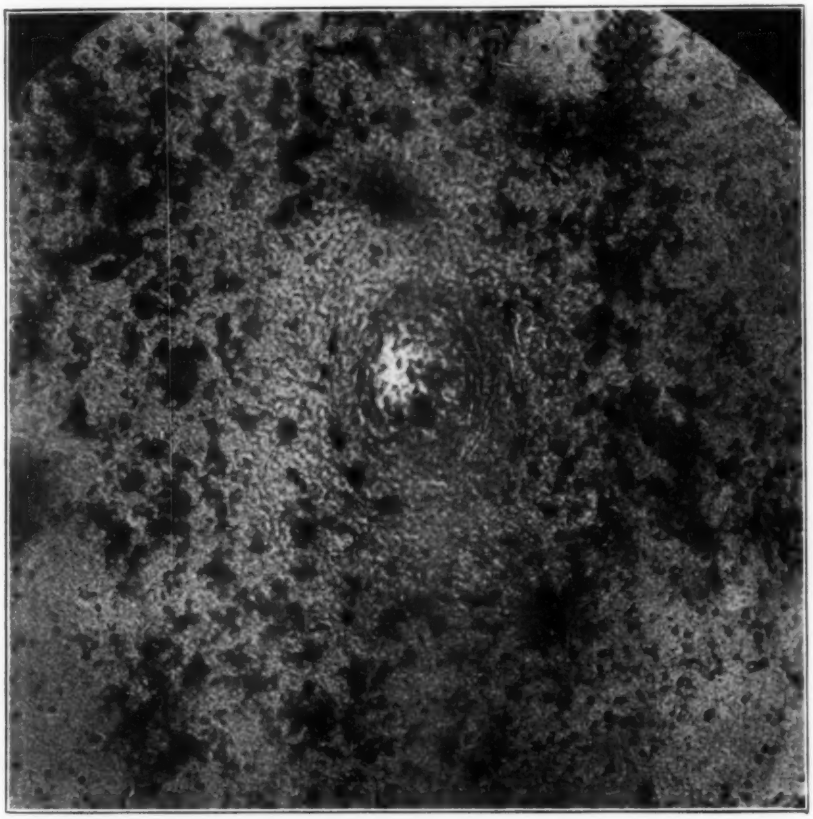

Fig. 3.—Photomicrograph of tuberculous nodule of brain, showing immense masses of tubercle bacilli surrounding arteriole and within its wall; × 103.

was that of Kuhne's suitable for celloidin sections, but the Ziehl-Neelson-Gabbet method was also used. Levaditi's method for demonstrating the *Treponema pallidum* in tissues was employed.

Cases in which findings similar to those just described have been found are few. The following have been selected as typical.

Greive[18] described a case of solitary tubercle in the right tegmentum, with degeneration in the fillet and superior peduncle of the cerebellum; occurring in a chronic tuberculous individual, male, aged 35, causing

paralysis of the right arm and leg. There were symptoms of brain tumor for nine months preceding death. Post mortem examination showed a chronic ulcerative tuberculosis of the lung, fatty liver and enlarged spleen. Examination of brain showed the meninges to be free from disease. The blood vessels in the right sylvian fissure showed extensive miliary and a few larger tubercles. The tumor in

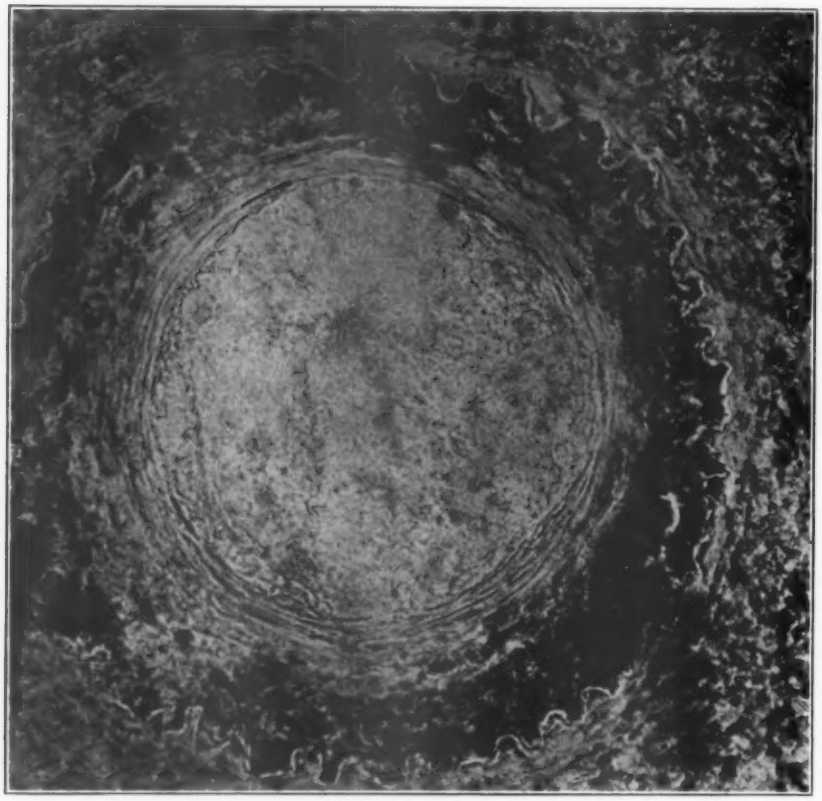

Fig. 4.—Photomicrograph of arteriole in tuberculous nodule of brain, showing endarteritis and tubercle bacilli within lumen; × 750.

the right tegmentum was of hazelnut size, its largest diameter being about 1.7 cm.; it showed central softening and was apparently made up of an aggregation of smaller foci fused together. It could easily be shelled out. The microscopic description of the tumor is very similar to the one just described. The blood vessels were everywhere extensively involved in an endarteritis and periarteritis and the walls were definitely diseased. The condition found in a small artery in the periphery of the growth is of interest. Innumerable masses of tubercle bacilli were found in its thickened walls as well as in the

tissues for a short distance surrounding it, while in other parts of the mass very few if any bacilli could be found.

N. Dalton[8] described a case of multiple tuberculous tumors of brain occurring in a child, 1 year of age. There were three tumors present, one in the pons, another at the back of the right internal capsule, and a third one in the cerebellum. There was no meningitis. Post mortem examination showed miliary tubercles present in nearly all organs. There was no history of tuberculosis or of syphilis in the family. The onset of symptoms followed a fall. There was paresis of both limbs and athetosis of the right arm. Otherwise no distinctive symptoms occurred. Death was apparently due to ulcerative stomatitis.

Renz[39] described a case of solitary tubercle of the left parietal lobe, the size of a billiard ball, occurring in an individual, 30 years of age. The patient was first seen with symptoms which pointed strongly to epilepsy. At that time tuberculous involvement of both apices of lungs was also found. With the exception of headache and vomiting, no new symptoms appeared. Post mortem examinations showed old healed tuberculous foci in both apices of the lungs, tuberculosis of right kidney and left suprarenal body, miliary tuberculosis of peritoneum covering the kidney and a solitary tubercle of left parietal lobe. The patient died at the end of one year.

J. L. Stevens[44] described a case of multiple tuberculous tumors in various parts of the brain, basal ganglia and cerebellum (from six to eight in all) occurring in a child 8 months old. The average size of the nodules was about that of a hazelnut. Clinically the symptoms were diverse and confusing "but in keeping with numerous tumors."

W. C. Chaffey[5] reports a case in a child 2 years old with caseous tuberculous deposit in the floor of the fourth ventricle, 5/8 by 3/8 inch, and also smaller nodules about the size of an acorn in each lateral surface of the cerebellum and one in the upper anterior border of the right side of the pons. There were symptoms of tumor for four months previous to death, namely, left ptosis, paresis of the left side of the face; both lower extremities were very weak; the upper extremities were normal, and both knee jerks were increased. During life no tuberculous disease could be discovered anywhere. At necropsy both lungs were studded with fine miliary tubercles, the bronchial glands were tuberculous, as was also the lower part of the left kidney.

F. X. Dercum[7] reported a large (7.5 cm. by 3.5 cm.) subcortical tuberculous tumor of the left occipital lobe, in a boy of 12 years of age, producing right sided hemiparesis and right homonymous hemianopsia, together with Wernicke's pupillary reaction as a distinct symptom. This tumor destroyed radiation of the optic thalamus, pressed

on the optic thalamus and corpora and probably caused slight pressure on the posterior limb of the capsule. Symptoms of tumor were present for at least one year preceding death.

J. S. Bristowe[2] reported several cases of tumors involving the parts in the neighborhood of the third and fourth ventricles and aqueduct of Sylvius, three of which are of the tuberculous type.

Case 1.—Child, aged 7 years, tuberculous tumor, size of a marble, of the corpora quadrigemina followed by tuberculous meningitis, paralysis of both third nerves and ptosis and tremors of head, neck, arms and legs. No fits. Sickness and headache absent until end. No optic neuritis. On necropsy pleurae were found adherent, and gray tubercles were scattered on the lung; larger tubercles occurred in the spleen.

Case 2.—Child aged 4 years. Tuberculous mass 1.5 cm. in diameter in third ventricle involving both thalami; also a smaller mass on internal surface of occipital lobe followed by tuberculous meningitis. Tremors and rigidity of arms and legs, drowsiness and irritability, optic neuritis and slight facial palsy were present. Convulsions, coma and elevation of temperature introduced a meningitis. No other tuberculous disease was found in the viscera, but patient had been operated on for scrofula of astragalus. Symptoms of tumor lasted for nine months.

Case 3.—Child aged 4 years. Nine months' duration. No tuberculous disease found during life. Large tuberculous mass size of tangerine orange in left lobe of the cerebellum; another small mass imbedded in the gray matter of orbital surface of the frontal lobe. Effusions of fluid into ventricle, inability to walk, tremors of arms and head, optic neuritis, and blindness, frequent epileptiform attacks, headache, etc. No meningitis was present. Glands at root of lungs were caseous and there was a pyelitis of both kidneys; otherwise no other lesions were found.

The most interesting observations in the case reported by us are the great masses of tubercle bacilli found in the vessel-walls and within the caliber of the vessels in the tubercles in the brain and bronchial lymph nodes. The disintegration of the caseating thrombi would result in throwing great masses of tubercle bacilli into the circulation. We have been unable to find in the literature the report of any case of this condition in which tubercle bacilli were found within the blood vessel. The only cases in which tubercle bacilli are described as having been found in masses about the vessels and in the vessel walls is that of Greive.[18] Our observation is therefore unique.

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4. Cerf: Deutsch. Arch. f. klin. Med., 1882, xxxi, 431. (Child, 2¾ years old. Miliary tuberculosis; one brain tubercle and circumscript tuberculous meningitis.)

5. Chaffey: Tr. Path. Soc. London, 1886, xxxvii, 63. (Child, aged 2 years, symptoms of tumor four months and one week; during life no tubercle bacilli discovered.)

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THE AMMONIA AND UREA CONTENT OF INFANTS' STOOLS

WITH A DESCRIPTION OF METHODS *

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Since bacteria, like other living cells, require a certain intake of nitrogenous material for the maintenance of the chemical structure of their protoplasm, and many of them are also able to utilize it for their energy needs, it is evident that the mediums in which they grow must contain end products of their nitrogen metabolism. Steps in the utilization of protein by bacteria are not in the gross dissimilar from those of the same process in the complex animal organism. They hydrolyze protein, so far as we know, in exactly the same manner as do the digestive ferments and are able ultimately to split off ammonia from the products of hydrolysis just as this is accomplished somewhere in the body. It is very interesting that besides ammonia another familiar end product of protein metabolism—urea—has been measured in appreciable amounts in cultures of bacteria which are normal or occasional inhabitants of the lower intestinal tract.[1]

The presence of ammonia in the stool may usually be demonstrated by adding water to a sample, making the mixture alkaline with potassium carbonate and then heating gently. The escaping ammonia may be detected by smelling, by the turning blue of a strip of moistened red litmus paper, or by noting the white deposit of ammonium chlorid on a glass rod which has been dipped in hydrochloric acid. The presence of urea is shown by an increase in the ammonia content after adding urease (see below). That all of the ammonia and urea in the stool is produced by bacterial proteolysis is certainly open to question. The intestinal ferments split ammonia from the "amid fraction" of the protein molecule. Whether the small amount thus produced is absorbed before reaching the stool mass in the large intestine is not known. There is also some evidence that ammonia is excreted to a slight extent into the upper part of the intestine and that in some conditions of disease, nephritis, for instance, the amount may be con-

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* From the wards of the Boston Floating Hospital and from the Children's Department of the Massachusetts General Hospital.

1. Kendall, Arthur I, and Walker, Arthur W.: Determination of Urea Nitrogen in Cultures of Certain Bacteria, Jour. Biol. Chem., August, 1913, p. 277.

siderable. On the other hand, Folin and Denis[2] by experiments with cats have demonstrated that ammonia is absorbed from the large intestine when it contains feces. They have also found that solutions of urea are rapidly absorbed from an empty large intestine.[3] Whether urea may under certain conditions be excreted into the intestine is not known. Although the balance between possible excretion and proved absorption of these substances is not known, it is probable that the amounts of ammonia and urea found in the stool represent fairly closely the extent, possibly not the full extent rather than the reverse, of bacterial proteolysis in the large intestine.

That total nitrogen determinations in the stool may contain an error due to loss of ammonia nitrogen from the specimen has been well recognized, and precautions are advised for the prevention of this during the collection, storage and drying of stools. Since the determination of nitrogen balance is often an essential point in metabolism experiments, and since the size of the error which may be caused by the loss of ammonia nitrogen is not at all definitely known, it has seemed to us worth while to determine the usual—and unusual—amounts of ammonia which may be found in fresh stools, the pace at which it increases while the stool is stored, and the amount which may be driven off during drying.

It was thought, moreover, that a simple method of determining the ammonia content of fresh stools might be useful in another direction. The necessary ability which bacteria have of metabolizing protein has been given the uncomfortable name "putrefaction." While the digestive ferments do not split protein further than the amino-acid stage, bacteria are able to carry the process to completion by decomposing these *Bausteine* of the protein molecule. From amino-acids of the aromatic type they produce substances—phenol, indol, skatol, and others—which if absorbed from the intestine are suspected of causing harm to the organism unless it protects itself by conjugating them with sulphuric or glycuronic acid and excreting them as inert esters—ethereal sulphates, chiefly—in the urine. (It should be mentioned here that although these derivatives of tyrosin, tryptophan, and phenylalanin are not produced by the digestive ferments, it is likely that they are formed within the body during the ultimate catabolism of these amino-acids.) As it would seem fair to take the ammonia content of fresh stools as an index of the extent of intestinal putrefaction, and as the method for determining ammonia in stools described below is rapid and simple, it is suggested that such determinations would be

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useful in the study of cases clinically labeled "intestinal auto-intoxication," "intestinal toxemia," etc.

The literature contains scant information as to the amount of ammonia found in fresh stools. Brauneck,[4] in 1886, reported the average amount of ammonia in stools from normal adults to be 0.151 per cent. of the dried stool, and that stools from nephritics contained on the average 0.343 per cent. He used the old Schlösing method for determination of ammonia in the urine. The stool sample was made acid with hydrochloric acid and dried. A hot water digest was made from the powdered stool, and lead acetate added. The filtrate, after setting free the ammonia in it by adding milk of lime, was placed in a flat dish under a bell-jar and over another dish containing fourth-normal sulphuric acid. After five days the ammonia taken up by the sulphuric acid was measured by titration. H. Bartling,[5] using undried samples in which he set the ammonia free by adding soda and collected it in tenth-normal hydrochloric acid by distilling under vacuum at 42 C., found in stools from an adult, on ordinary diet, who was normal except for anemia, an ammonia content of 0.398 per cent. of dried substance. After the addition to the diet of 300 gm. calf's thymus the ammonia rose to 0.625 per cent. The total nitrogen in the stool, however, was not increased, so that the ammonia nitrogen in per cent. of total nitrogen rose from 4.9 per cent. when on ordinary diet to 8.3 per cent. when extra protein was given. Both these workers regarded the ammonia found in feces as excreted from the wall of the intestinal tract.

METHODS OF PROCEDURE

Method of Collecting Specimens.—The baby was placed on a Bradford frame in the manner which has been described by Talbot.[6] The stools were collected for a period of twenty-four hours, beginning at 9 o'clock in the morning. The receiving cup was inspected at hourly intervals by a nurse, and when found to contain feces was removed and placed directly on ice, another cup taking its place in order to keep the stools as fresh as possible. The urine was collected separately. The determinations for total nitrogen, ammonia, and urea nitrogen were carried out within a few hours after completion of the twenty-four hour collection period.

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Method of Analysis.—Total nitrogen: The following modification of Folin's micro-Kjeldahl method for total nitrogen in the urine was devised and found to give very satisfactory results. The twenty-four hour specimen of stool is placed in a weighed dish and thoroughly mixed by stirring and rubbing with a spatula, water being added if necessary. Its weight is then determined and a 2 to 5 gm. sample weighed out in a small evaporating dish, over this is poured from 60 to 70 c.c. 20 per cent. sulphuric acid and the dish placed on the water-bath for from one-half to one hour. The resulting digest is then transferred to a 100 c.c. volumetric flask, cooled and made up to the mark with 20 per cent. sulphuric acid. From this two 5 c.c. samples are pipetted into large Jena glass test tubes, and 1 gm. potassium sulphate, two drops 10 per cent. copper sulphate and a small pebble added to each. No sulphuric acid need be added as enough is contained in the samples. The tubes are then placed over micro-burners and from this point the determination is carried on as in the method for urine. The digestion mixture should be boiled carefully at first in order to avoid driving it very far up the sides of the tubes. Digestion will be found to be complete in twenty or twenty-five minutes. One point which is not quite satisfactory must be mentioned. The preliminary digest often contains minute particles which tend to settle, and for this reason the mixture must be shaken immediately before pipetting the samples. All danger of error may be avoided by carrying out the preliminary digestion in a small Kjeldahl flask over a flame, and this plan must be followed when the stool contains casein curds. It was found, however, that duplicates from the usual type of stool, digested on the water-bath, were in close agreement, and also agreed with the result obtained by carrying through a sample by the regular Kjeldahl method. While no essential advantages can be urged, it will be found that as compared with the usual way of doing Kjeldahls, this modification is shorter and less cumbersome, and where other colorimetric nitrogen determinations are being carried out at the same time, it fits conveniently into laboratory routine. The micro-method can also be very conveniently used for determination of total nitrogen in milk, in which case no preliminary digestion is necessary. The milk is simply diluted until 5 c.c. will contain about 1 mg. of nitrogen.

Ammonia Nitrogen: At the suggestion of Dr. Denis, the Folin method for ammonia in the urine was used. Folin and Denis have employed it occasionally for the determination of ammonia in feces in the course of animal experiments. The steps are very simple. A 1 or 2 gm. sample of well mixed stool is weighed out on a small bit of oiled paper. The paper and sample are then dropped into a large test-tube, and about 5 c.c. of water added. If the stool is fluid, it is

weighed in a small porcelain dish and rinsed into the test tube with 5 c.c. of water. A few drops of kerosene and 1 or 2 c.c. of a solution containing 15 per cent. potassium oxalate and 15 per cent. potassium carbonate are then added and the determination carried on as in the method for urine, except that the air current is allowed to run twenty minutes instead of ten. All determinations were made in duplicate and the results averaged. In most of the stools examined the agreement between duplicates was within 3 per cent. Considering that the samples were small and weighed separately on an ordinary inexpensive balance, such a result is satisfactory, and is interesting as showing the very uniform composition of the usual type of infants' stool. Stool containing casein shreds and curds gave a difference between duplicates of from 5 to 10 per cent.

Urea Nitrogen: A 1 or 2 gm. stool sample was placed in a large test-tube with 5 c.c. of water, 1 c.c. of 15 per cent. "Arlco-urease" and a few drops of kerosene. It was then stoppered and connected with the compressed air tap and allowed to stand thirty minutes at room temperature with a rather slow air current passing through it, for the purpose of breaking up the stool sample. A receiving tube, containing 2 or 3 c.c. of tenth-normal hydrochloric acid, was of course in position. One or 2 c.c. of the potassium oxalate and carbonate solution were then added through the inlet tube and the blowing resumed vigorously for twenty minutes. From the ammonia nitrogen obtained was subtracted that found in a sample similarly prepared, but without the addition of urease. The difference was regarded as urea nitrogen. That this is the case cannot be accepted without reservation. Marshall[7] has shown the specific action of urease on urea in the urine. That this holds true among the many nitrogen-containing compounds present in feces is probably the case, but would be an extremely difficult point to prove. Duplicate determinations for urea were found to agree with the same closeness as in the case of ammonia.

RESULTS AND DISCUSSION

The Usual Ammonia Content of Infants' Stools and Its Relation to the Amount of Protein in the Diet.—In Table 1 are given thirty-seven determinations of total nitrogen and ammonia nitrogen from twenty-four hour stool specimens. It being realized that the day-to-day amount of stool passed must vary considerably, the amounts of nitrogen and ammonia per gram of dried stool are also given. These later figures probably afford a better basis for comparison but are not entirely satisfactory because of being affected by the variable amount

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TABLE 1.—THIRTY-SEVEN DETERMINATIONS OF TOTAL NITROGEN AND AMMONIA NITROGEN FROM TWENTY-FOUR-HOUR STOOL SPECIMENS

Number	Gm. Protein per Kilo in Food	Nitrogen in Stools		Ammonia N. in Stools		Ammonia N.: Total N. Per Cent.
		Total for 24 Hours mg.	Per Gm. Dried Stool mg.	Total for 24 Hours mg.	Per Gm. Dried Stool mg.	
13.3	1.2	102	53	5	2.4	5.0
11.4	1.7	218	53	16	3.9	7.9
23.2	1.8	191	24	21	2.6	11.0
19.1	2.0	488	44	23	2.1	4.7
5.1	2.3	298	25	29	2.5	9.7
9.2	2.4	750	54	47	3.4	6.3
13.1	2.9	360	63	19	3.3	5.3
10.2	2.9	203	24	23	2.7	11.3
1.3	2.9	417	39	39	3.6	9.4
24.1	3.6	216	42	16	3.1	7.4
10.1	3.8	480	38	61	4.8	12.7
6.1	4.1	230	40	25	4.4	11.9
25.1	4.2	312	58	31	5.7	10.0
1.1	4.5	347	26	16	1.2	4.6
18.1	4.6	501	35	43	3.0	8.6
5.2	4.4	427	39	53	4.8	12.4
5.3	4.8	492	38	40	3.1	8.1
17.1	5.0	215	67	17	5.3	1.9
24.2	5.0	384	42	40	4.3	10.4
24.3	5.0	653	53	53	4.3	8.1
11.1	5.2	625	41	49	3.2	7.9
8.1	5.2	417	37	46	4.1	11.0
4.1	5.3	118	29	3	0.7	2.5
3.1	5.3	447	27	67	4.0	15.0
13.2	5.4	305	46	35	5.2	11.5
19.2	5.5	259	67	33	9.9	12.7
5.4	5.9	640	46	61	4.4	9.5
16.1	6.1	202	37	17	3.1	8.4
11.3	6.5	648	43	54	3.6	8.3
11.2	6.6	710	50	72	5.0	10.1
5.5	7.1	1,159	65	85	4.7	7.3
20.2	8.0	451	68	55	8.3	12.2
10.4	8.6	298	93	37	11.6	12.4
10.3	8.6	170	35	11	2.2	6.5
23.3	8.9	571	65	33	3.8	5.8
23.1	9.5	1,436	126	65	5.7	4.5
23.2	9.5	628	72	63	7.2	10.0

of fat in the stool. The data are arranged with reference to an increasing protein intake. The babies supplying these specimens were all practically normal, that is, they came to the hospital without history of serious nutritional disturbance and when their food was regulated they gained weight in the normal way. Four of them, Nos. 20.2, 23.1, 23.2, and 24.3, were given buttermilk, and three, Nos. 16.1, 17.1, 19.2, received Eiweiss milk. The rest were on ordinary modifications of cow's milk. In a good many instances several specimens were obtained from the same baby, as is indicated in the number column by recurrence of the same figures in the integral part of the numbers. The age of the babies was between 2 and 10 months. A glance at the table will show that the ammonia content is widely variable but tends to grow larger as the protein intake is increased.

TABLE 2.—Averages of Groups Indicated in Table 1

No. Observations	Protein Gm. per Kilo in Food	Gm. Carbohydrate per Kilo in Food	Nitrogen in Stools		Ammonia N. in Stools		Ammonia N.: Total N. Per Cent.
			Total mg.	Per Gm. Dried Stool mg.	Total mg.	Per Gm. Dried Stool mg.	
6	1.9	13	341	42	23	2.8	7.4
11	3.9	13	362	40	33	3.6	9.3
13	5.5	11	432	45	42	4.4	9.5
7	8.6	11	673	76	50	6.2	8.4
37	5.0	12	443	52	38	4.2	9.0

This is very evident in Table 2, in which the groups indicated in Table 1 are averaged. In the first three groups the total nitrogen per gram dried stool is about the same. Apparently as much as 5.5 gm. protein per kilo is nearly as completely utilized as 1.9 gm. It must also be admitted that the increase in ammonia content as more protein is utilized could be interpreted as evidence that the excretion factor may be larger than we are assuming. Except when buttermilk or Eiweiss milk was given, the babies received a usual amount of carbohydrate. The average amount in grams per kilo is given in this table, and will be seen to be nearly the same for the four groups. According to the views of Kendall[8] based on culture-flask experiments, if a sufficient amount of carbohydrate is available bacteria will utilize it for their vegetative needs and metabolize protein only to the extent necessary for the maintenance of their chemical structure, no matter how

8. Kendall, Arthur I., and Farmer, Chester J.: Studies in Bacterial Metabolism, viii, Jour. Biol. Chem., October, 1912.

much of it may be at hand. Interpreting the results presented here in this light, we must assume that when the usual amount of carbohydrate is fed babies, the portion of it escaping absorption and reaching the large intestine is altogether inadequate to supply fuel for the vegetative functions of the bacteria. Kendall's experiments are quite convincing on the point that bacteria prefer to metabolize carbohydrate up to a certain point, rather than the nitrogenous substances contained in bouillon, but whether enough carbohydrate can be given a baby, without overtaking its tolerance for it, to bring about in the lower bowel an imitation of his culture-flask conditions, seems to us problematical. Average figures for the thirty-seven specimens are also presented. It will be seen that the average ammonia nitrogen content was 38 mg. or 4.2 mg. per gram of dried stool. The highest ten in the series gave an average of 64 mg. or 6.8 mg. per gram dried stool, and

TABLE 3.—AVERAGE AMMONIA CONTENT FOR THREE GROUPS ARRANGED ACCORDING TO THE NUMBER OF STOOLS PASSED

No. Observations	Gm. Protein in Food per Kilo	Number of Stools 24 Hours	Nitrogen in Stools		Ammonia N. in Stools		Ammonia N.: Total N. Per Cent.
			Total mg.	Per Gm. Dried Stool mg.	Total mg.	Per Gm. Dried Stool mg.	
9	5.3	1	321	46	27	3.3	7.6
12	5.4	2– 4	322	55	34	5.5	10.0
15	4.9	5–10	608	48	48	3.7	8.6

the lowest ten 14 mg. or 2.3 mg. per gram dried stool. The average for ammonia nitrogen in per cent. of total nitrogen was 9.0, and in per cent. of dried stool substance 0.42.

Ammonia Content in Relation to Number of Stools Passed.—The results were rearranged in three groups according to the number of stools in twenty-four hours. The averages for these groups are given in Table 3. The amount of protein in the food was, in average, about the same in the three groups. It will be seen that the ammonia content increases as the stools become more frequent. That the ammonia per gram dried stool is not higher in the third group than in the second is undoubtedly due to the large amount of unutilized fat in the stools when they are very frequent. The increase in the extent of putrefaction is probably due to the fact that more nitrogenous material escapes absorption, and to the better opportunity for bacterial activity afforded by the more fluid texture of the stools. That constipated stools contain the least ammonia is interesting, because of the prevailing suspicion that constipation is an ally of intestinal putrefaction.

TABLE 4.—Four Cases of Stools with Unusually High Ammonia Content

Diagnosis	Food	Gm. Protein per Kilo in Food	Number Stools 24 Hours	Nitrogen in Stools		Ammonia N. in Stools		Ammonia N.: Total N. Per Cent.
				Total mg.	Per Gm. Dried Stool mg.	Total mg.	Per Gm. Dried Stool mg.	
Indigestion	Buttermilk ...	8.0	6	1,556	98	237	15.0	15.2
Malnutrition ..	Eiweiss	8.2	3	620	55	150	13.3	24.0
Atrophy	Buttermilk ...	5.4	6	770	87	168	19.0	22.0
Atrophy	1.5-6-1.2	5.8	11	1,022	42	148	6.0	14.5

Stools with Unusually High Ammonia Content.—Four such were encountered—see Table 4. Three were from babies in an advanced stage of malnutrition, the large amount of unutilized protein reaching the lower part of the intestine, as shown by the high nitrogen content of the stools, is probably a sufficient explanation of the unusual degree of putrefaction. It is worth noting, to the discredit of its alleged efficacy in reducing intestinal putrefaction, that two of these babies were receiving buttermilk.

Increase in Ammonia in Stools on Standing.—The extent of this is shown in Table 5. It will be seen there that the amount of ammonia

TABLE 5.—INCREASE IN AMMONIA IN STOOLS ON STANDING

NH_3 Nitrogen Fresh Stools		NH^3 Nitrogen 24 Hours After Collection		NH_3 Nitrogen 48 Hours After Collection	
Total mg.	Per Cent. Total Nitrogen	Total mg.	Per Cent. Total Nitrogen	Total mg.	Per Cent. Total Nitrogen
STOOLS STANDING AT ROOM TEMPERATURE					
26	5	...	...	41	8
55	12	87	19	110	24
71	5	167	12	212	15
40	10	67	17	...	...
21	11	...	...	42	22
31	10	...	...	74	24
STOOLS STANDING IN ICE-BOX					
26	5	...	...	31	6
16	7	...	...	17	8
54	8	...	...	80	12
33	6	...	...	35	6

found in stools standing at room temperature for forty-eight hours was two or three times as much as the stools when fresh. Stools stored for the same length of time in the ice-chest showed only a very slight increase in the amount of ammonia contained.

Loss of Ammonia from Stools while Standing.—Twenty gram samples from six stools were placed in 200 c.c. Erlenmeyer flasks. Each flask was closed by a rubber stopper through which passed two pieces of glass tubing. The outer ends of the tubing were closed by slipping rubber tubing over them and clamping it. The samples were left standing at room temperature for forty-eight hours. The flasks were then connected with the compressed-air tap, and the air inside them driven out, and into a test tube containing a few cubic centimeters

of tenth-normal hydrochloric acid. Three of the stools were alkaline and three acid. On testing, in each case, the hydrochloric acid in the receiving tube with Nessler's reagent it was found that from two of the alkaline stools a trace of ammonia—not measurable in the colorimeter—had been collected. No ammonia had been given off from the other four specimens. Evidently then there is not much danger of loss of ammonia from stools stored in closed vessels.

Loss of Ammonia from Stools during Drying.—A ten gm. sample of fresh stool was spread over the bottom of a 200 c.c. Erlenmeyer flask, provided with a rubber stopper carrying inlet and outlet tubes as described as above. The flask was then placed on the water-bath

TABLE 6.—Loss of Ammonia from Stools During Drying

Reaction	Total Nitrogen in Fresh Stool mg.	Ammonia Nitrogen in Fresh Stool mg.	Ammonia Nitrogen Collected During Drying		Nitrogen in Dried Stool + Ammonia Nitrogen Collected mg.	Ammonia Nitrogen in Dried Stool mg.
			Total mg.	Per Cent. Total Nitrogen		
Alk.	202	17	17	8.4	211	
Alk.	1,556	237	341	21.9	1,562	
Ac.	417	39	34	8.2	401	
Ac.	710	72	89	12.5	713	
Ac.	501	43	9	1.8	498	
Alk.	800	71	62	7.8	780	6
Alk.	648	54	83	12.8	642	9
Ac.	488	23	6	1.2	486	18
Alk.	215	17	36	16.8	193	
Ac.	295	50	43	14.6	...	8
Ac.	620	150	89	14.4		
Alk.	451	55	67	14.9		
Alk.	628	63	72	11.5		
Alk.	1,436	65	42	2.9		
Alk.	653	53	66	10.1		
Alk.	203	23	28	13.8		5

and connected with the compressed-air tap. A slow current of air was allowed to pass through the flask, and then through the tenth-normal hydrochloric acid in a receiving tube, while the stool sample was drying. The ammonia collected in the receiving tube was then measured by adding Nessler's, making up to an appropriate volume and reading in the colorimeter. The results of a series of these experiments are given in Table 6. The figures for total nitrogen and ammonia nitrogen are for the twenty-four hour specimen. It was found that the acid stools lost a large part of their ammonia, and that from the alkaline stools more ammonia was usually collected than the stools contained before drying. That the cause of this ammonia

production during drying is bacterial activity does not seem likely as the time required was short—from one and a half to two hours—and the decreasing moisture in the stool unfavorable to bacterial activity. It would seem more probable that the extra ammonia is derived from some nitrogenous substance or substances decomposed by the heat and drying. It is possible that its chief source may be urea. The urea contained in two of the specimens in the series was measured and found to be 20 and 16 mg. The extra ammonia collected from these two was 12 and 13 mg. A few determinations, given in the last column in the table, showed that some ammonia was still contained in the dried stool. A comparison of the figures in column 6 in

TABLE 7.—UREA NITROGEN IN FIFTEEN TWENTY-FOUR-HOUR STOOLS

Total Nitrogen mg.	Ammonia Nitrogen mg.	Urea Nitrogen Total mg.	Urea Nitrogen per gm. Dried Stool mg.	Urea N.: Total N. Per Cent.
170	11	8	1.0	4.7
298	37	11	3.4	3.7
295	50	13	1.9	4.4
268	7	15	4.4	5.6
488	23	10	0.9	2.0
259	33	4	1.0	0.6
620	150	24	2.1	3.9
451	55	20	3.0	4.4
1,436	65	12	1.1	0.8
191	21	9	1.1	4.7
571	33	15	1.7	2.6
216	16	9	1.8	4.2
384	40	12	1.3	3.1
653	53	16	1.3	2.5
312	31	16	3.0	5.1

this table with those in column 2 will show fairly well the degree of accuracy, attained by the micro-Kjeldahl method for total nitrogen.

From these experiments we conclude that there is practically no danger of loss of ammonia nitrogen from stools during collection and storage, unless the specimen is allowed to lose moisture; but that during drying a very serious loss may occur, often as much as from 15 to 20 per cent. of total nitrogen from fresh stools, while from stools left standing for two days at room temperature one half of the total nitrogen may escape measurement. For the prevention of this loss the addition of preservatives is obviously inefficient. The only measure which has a sound basis is the addition of non-volatile acid with the object of fixing the ammonia. This was tested and found to be nearly always completely efficient provided enough acid was thoroughly mixed in the stool. It should be remembered that the texture of the stool

substance offers obstacles to diffusion and that as its particles dry, ammonia escaping from them may in the absence of water escape contact with the acid. Adding a crystal or two of oxalic acid or a few drops of weak hydrochloric acid is not by any means sufficient, even though this may make the stool acid to litmus. Five c.c. of 2 per cent. hydrochloric acid well mixed with 10 gm. of stool was found to prevent any loss of ammonia from stools of the usual type but not from those containing casein shreds and curds. Since the danger of losing nitrogen is entirely avoided by making the determinations from an undried sample we prefer the wet method. We are convinced that as even a sample is obtained from the fresh as from the dried and powdered stool in most instances. There is certainly less chance for error in weighing a 5 to 10 gm. sample of fresh stool than comes into the process of grinding up the dried stool, determining its weight and

TABLE 8.—INCREASE IN UREA IN STOOLS STANDING SEVERAL DAYS AT ROOM TEMPERATURE

Urea N in Fresh Stool mg.	Urea N 24 Hours After Collection mg.	Urea N 48 Hours After Collection mg.	Urea N Three Days After Collection mg.	Urea N Five Days After Collection mg.
10	..	..	7	33
20	5	12	..	..
14	5	..	93	122
9	..	14	..	28
12	..	21	..	..
16	..	26	..	..

then weighing out a 0.25 to 0.50 gm. sample of the dusty hygroscopic powder. Moreover, by the former procedure one escapes the very frequent annoyance of encountering stools which, because of the neutral fat they contain, will not powder, unless they are dried with sea-sand or plaster of Paris, thus introducing another source of error. Even in the case of stools containing much mucus or casein curds we prefer to make a preliminary digest of a large sample and determine the nitrogen in a measured portion of this rather than trust to the evenness of a sample of stool powder.

The Urea Content of Stools.—Urea nitrogen was determined in fifteen twenty-four hour specimens from practically normal babies. The amounts found are given in Table 7. The average for the fifteen stools was 13 mg. or 2.0 mg. per gram dried stool. The average in per cent. of total nitrogen was 3.5. As may be seen in the table, the proportionate relationship between urea nitrogen and ammonia nitro-

gen is widely variable. Whether or not some of the urea found in stools was excreted by the intestinal wall, we are unprepared to say. That the intestinal flora are capable of producing urea is evident from an increase in the amount of it found in stools standing several days at room temperature. Determinations showing this are given in Table 8.

Thanks are due to Dr. F. B. Talbot for providing every facility for obtaining material for these experiments from his wards on the Boston Floating Hospital and in the Massachusetts General Hospital, and for his encouraging interest in the results; to Dr. W. Denis for many useful suggestions in the laboratory, and to Miss Visel of the Floating Hospital and Miss Richardson of the Massachusetts General Hospital for their extremely careful supervision of the collection of specimens.

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FORMS OF NITROGEN IN THE STOOLS OF INFANTS *

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It has for some time been recognized that the *amount* of nitrogen lost in the stools of infants is rarely of great importance from the point of view of nutrition. The percentage of the intake which escapes absorption seldom constitutes a serious loss. It seemed possible, however, that significant information concerning the manner in which the alimentary tract handles the food proteins under varying conditions might be obtained by a study of the relative *proportions* of the fecal nitrogen which are excreted in the form of protein, and of amino acids and ammonia, the two chief final split products of protein. When proteins are digested under aseptic conditions by the intestinal enzymes, trypsin and erepsin, amino acids are the main products, and the amount of ammonia is relatively small. Putrefactive bacteria, on the other hand, are capable of changing a large percentage of the nitrogen of proteins and amino acids into ammonia. Consequently an increase of ammonia in the fecal nitrogen at the expense of the amino acids might serve as an indication of the activity of putrefactive bacteria. A relatively high proportion of amino acids, on the other hand, might indicate incomplete absorption of the protein digestion products, despite complete digestion and absence of putrefaction.

Our results failed to show any constant relations between the clinical condition of the patients and the distribution of the fecal nitrogen among the three forms discussed above. The data obtained are, however, not without interest in their revelation of the form in which the unabsorbed nitrogen leaves the alimentary canal, particularly as previous information on this point has been very meager.

METHODS

The method of collection made it almost impossible for a urine contamination to occur without our knowledge. The stools were used either when freshly voided or were placed at once in an ice-box to

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* From the Laboratories of the Rockefeller Institute and the Babies' Hospital.

* The observations reported here were begun in the fall of 1913, and have since been continued at intervals as cases suitable for study have presented themselves.

avoid any further decomposition. Forty to 50 gm. of the fresh feces were emulsified in water and diluted to 200 c.c. The mixture was made homogeneous by grinding in a mortar and aliquot portions were taken for the following determinations.

Total Nitrogen.—Five c.c. were used for a Kjeldahl determination.

Ammonia.—Ten c.c. were placed in a 100 c.c. test tube, 4 to 5 gm. of potassium carbonate, with 2 drops of octyl alcohol to prevent foam, were added, and the ammonia was determined by aeration into fiftieth-normal acid by Folin's method.

Urea.—Ten c.c. were placed in a 100 c.c. test tube with 5 c.c. of 0.6 per cent. KH_2PO_4 and 1 c.c. of 10 per cent. "Arlco-urease."[1] After fifteen minutes, allowed for hydrolysis of the urea, from 8 to 10 gm. of potassium carbonate were added and the ammonia aerated into fiftieth-normal acid. The increase in ammonia resulting from the action of the urease represents the urea nitrogen. The accuracy of the method under the conditions described was shown by control analyses on ten specimens of feces. Known amounts of urea were added to measured portions of the emulsified feces. In every case the determinations showed an increase in urea equal to the amount added.

Free Amino Acid Nitrogen.—To 100 c.c. of emulsion about 0.5 gm. of charcoal was added, and the solution was filtered without washing the residue. The volume of the filtrate was measured, and to it 10 per cent. metaphosphoric acid was added until all the protein was precipitated. The volume of metaphosphoric acid solution required was 15 c.c. or less. After a half hour had been allowed for complete precipitation of the proteins, the mixture was made up to a convenient volume, made alkaline by addition of powdered calcium hydroxid, and filtered. The filtrate was free from protein, as indicated by the biuret test. The filtrate was measured and evaporated to dryness on a water bath. During evaporation the solution, made alkaline by the above mentioned addition of lime, entirely loses its ammonia. The foregoing treatment therefore frees the solution of both protein and ammonia. The dry residue was taken up with 1 c.c. of 50 per cent. acetic acid and washed into a 10 c.c. measuring flask with small portions of water. Two c.c. portions were used for determination of amino nitrogen by the nitrous acid method.[2]

Total Amino Acid Nitrogen after Hydrolysis of the Proteins.—Ten c.c. of the feces suspension were mixed with 10 c.c. of concentrated hydrochloric acid, and the solution, in a loosely stoppered tube, was immersed in a water bath at 100° for twenty-four hours. This

1. Van Slyke and Cullen: A Permanent Preparation of Urease and Its Use in the Determination of Urea, Jour. Biol. Chem., 1914, xix, 211.
2. Van Slyke: Jour. Biol. Chem., 1913, xvi, 121.

assures practically complete hydrolysis of the proteins present.[3] The solution was evaporated to dryness on the water bath to remove the hydrochloric acid. The residue was taken up with water, made alkaline with calcium hydroxid, and filtered. The alkaline filtrate was concentrated to dryness to remove the ammonia. It was then redissolved with the help of 1 c.c. of 50 per cent. acetic acid; the solution was made up to 25 c.c., and 2 c.c. portions were taken for determination of amino nitrogen.

Acidity.—An aliquot part, usually 25 c.c., of the suspension of feces was titrated with tenth-normal sodium hydroxid, phenolphthalein being used as indicator. The method is, of course, a crude one, but yields comparable results.

DISCUSSION OF RESULTS

As the accompanying tables show, the proportion of the total nitrogen in the form of NH_2 of the *free amino acids* varies greatly, the range being from 1.8 per cent. to about ten times as much—17.6 per cent. The present data show no factor in the diet or condition of the patients which has an entirely constant effect on the proportion of nitrogen passing unabsorbed through the intestine either as ammonia or free amino acids. The more nearly normal babies showed low amino acid nitrogen, however, and those suffering from diarrhea generally showed the higher figures. None of the three breast-fed infants was among those eliminating a large proportion of amino acid nitrogen, and the only baby in the series that was both normal and breast-fed showed next to the lowest proportion of amino acid nitrogen in the feces. In the case that was followed for seven successive days (Table 2) it was noted that when the stools became loose the proportion of amino acids was increased, and vice versa. As a rule, to which there are exceptions, it appears that in the other cases also the more watery the stools the larger is the proportion of their nitrogen in the form of amino acids. Apparently the quicker passage through the alimentary tract results in a less complete absorption of the final products of protein digestion, viz., the amino acids.

In contrast to the wide variations above mentioned in the nitrogen of the free amino acids, the nitrogen of the *amino acids present after hydrolysis of the protein* was quite constant at from 43 to 55 per cent. of the total nitrogen. In a few exceptions this percentage was necessarily lower because the ammonia was so high. Most animal proteins when hydrolyzed show on the average about 75 per cent. of their nitrogen as free NH_2, the other 25 per cent. being amid nitrogen and

3. Van Slyke: Conditions for the Complete Hydrolysis of Proteins, Jour. Biol. Chem., 1912, xii, 295.

TABLE 1.—FORMS OF—

No.	Age in mos.	Condition	Food †
1	5¾	Convalescent from diarrhea and wasting.	2-4-1.8 with barley flour.
2	4	Normal	Breast milk
3	18½	Drowsy and feverish, improving very slightly.	Soft diet; egg, bread, cereal, sauce, milk.
4	5½	Convalescent from diarrhea and wasting.	2-4-1.8 with barley flour, and malt soup extract.
5	8¾	Convalescent from diarrhea.	Protein milk
6	19	Feverish, with slight cough.	3-3.4-2.8 and cereal.
7	11	Nearly normal after acute indigestion.	Protein milk with barley flour.
8	7	Convalescent from enterocolitis.	1.4-1.75-1.2 (from condensed milk boiled with sodium bicarbonate) with cane sugar and barley flour.
9	6¼	Slowly convalescent from wasting, diarrhea and vomiting.	1.6-2-1.4 with barley flour (from condensed milk cooked with sodium bicarbonate).
10	5¾	Convalescent from diarrhea and wasting.	2-4-1.8 with barley flour and malt soup extract.
11	14¾	Slowly convalescent from diarrhea and wasting.	Protein milk
12	7	Convalescent from diarrhea.	Protein milk with barley flour and malt soup extract.
13	1	Congenital malformation of heart.	1.4-1.5-1.2, from condensed milk and boiled with sodium bicarbonate.
14	6¾	Convalescent from diarrhea and vomiting.	Protein milk with barley flour. Breast milk X3.
15	5	Marasmus, gaining slightly.	Breast milk.
16	3	Marasmus, gaining slightly.	Breast milk.
17	3	Marasmus, gaining slightly.	Breast milk.
18	5½	Convalescent from diarrhea and wasting.	2-4-1.8 with barley flour and malt soup extract.
19	5½	Convalescent from diarrhea and wasting.	2-4-1.8 with barley flour and malt soup extract.
20	7	Improving from diarrhea.	Protein milk with barley flour and malt soup extract.
21	5½	Convalescent from diarrhea and wasting.	2-4-1.8 with barley flour and malt soup extract.
22	25½	Suspected tuberculosis. Gaining weight.	House diet: scraped meat, potato, bread, cereal, sauce, milk.
23	1½	Gaining slightly from wasting and vomiting.	1.4-1.75-1.2 with barley flour (from condensed milk and boiled with sodium bicarbonate).
24	6¾	Slowly convalescent from wasting, diarrhea and vomiting.	1.6-2-1.4 changed to 1.8-2.5-1.6 with malt soup extract.
25	5½	Convalescent from diarrhea and wasting.	2-4-1.8 with barley flour and malt soup extract.
26	4¾	Improving from diarrhea.	Protein milk with cane sugar.
27	19¼	Convalescent from bronchopneumonia.	Milk, two parts: water, one part.
28	19½	Convalescent from bronchopneumonia.	Soft diet; milk, egg, bread, sauce, cereal.
29	8	Convalescent from diarrhea.	1.4-1.75-1.2 from condensed milk and boiled with sodium bicarbonate and barley flour. Breast milk X4.
30	12	Convalescent from pneumonia.	3-5-2.8 with beef juice, cereal, orange juice and sweet wine of iron.
31	7½	Improving slightly from diarrhea and vomiting.	1.8-2.25-1.6 from condensed milk and boiled with sodium bicarbonate and cane sugar and barley.
32	12	Convalescent from pneumonia.	3-5-2.8 with beef juice, cereal, orange juice and sweet wine of iron.
33	12	Convalescent from pneumonia.	3-5-2.8 with beef juice, cereal, orange juice and sweet wine of iron.
34	5	Marasmus improving.	Same as No. 35.
35	5¼	Convalescent from bronchopneumonia.	1-5-2
36	16¼	Physically normal, mentally deficient.	2.6-5-2.4 and cereal.

* The cases are arranged in order corresponding to the free NH_2 figures, the lowest being given first.

† The figures for the food formulas are given in the order, per cent. of fat, per cent. of milk sugar, per cent. of protein.

—Nitrogen in Infants' Stools *

Stools	Gm. Total N per Gm. Dried Wt.	Per Cent. of Total N. as			Acidity c.c. N/10 NaOH
		Free NH_3	NH_3 After Hydrolysis	Free NH_3	
Formed, hard and dry.....	.071	1.8	46.9	8.1	0
Watery in part; part pasty, greenish, sour. Soft curds.	.046	2.7	49.7	5.2	15.16
Constipated, formed and hard....	.055	2.9	55.1	7.7	1.84
Mostly formed, hard and soapy in part.	.054	3.3	49.7	3.6	2.57
Formed. Traces of mucus.....	.058	3.4	52.4	10.0	3.60
Constipated, formed and hard....	.064	3.7	52.0	18.8	3.05
Part formed, in part smooth and pasty.	(.05)	4.0	.....	12.0	.....
Mostly formed, part pasty and smooth.	(.05)	4.0	.....	13.0	2.70
Very watery. Mucus and soft curds.	.046	4.6	50.4	10.6	1.88
Mostly formed, soft and granular.	.073	4.8	43.4	6.9	3.13
Part formed, part pasty. Large curds.	.049	4.9	51.8	27.9	2.49
Part watery, part pasty, fermentative, acid. Mucus and curds.	.066	5.2	34.9	.....	4.65
Very watery. Mucus and large soft curds.	.11	5.8	25.3	9.7	11.28
Watery, large soft curds.....	.059	5.8	31.6	37.3	11.75
Part formed, part thin, pasty....	.046	6.2	50.6	7.8	14.59
Watery, greenish, sour. Fat curds.	.065	6.3	47.6	5.9	9.65
Watery, greenish, sour. Fat curds and mucus.	.059	6.4	47.4	3.8	10.61
Watery and soft pasty, sour. Large soft curds.	.051	6.6	47.7	7.6	7.95
Part formed, granular. Fine white curds.	.058	6.8	52.5	4.3	3.97
Part formed, part thin, pasty, acid, fermentative, mucus.	.050	6.8	.....	.....	9.82
Soft, pasty, granular. Soft curds.	.051	7.2	50.0	5.2	7.92
Thin pasty, oily-appearing.....	.035	7.6	47.8	7.0	12.60
Watery, full of fine white curds. Mucus.	.052	7.7	36.9	23.1	5.15
Part formed, part watery. Soft curds and mucus.	.071	8.0	41.8	29.2	.66
Part formed, granular. Fine white curds.	.053	8.4	49.0	4.7	3.74
Porridge-like, greenish.....	.029	9.0	50.4	19.5	7.37
Mostly formed. Mucus.....	(.05)	9.0	.....	13.0	2.60
Pasty, granular, mucus.....	.057	9.6	48.8	15.0	8.20
Watery, greenish, soft curds and mucus.	.065	10.2	55.5	7.6	9.30
Watery, acid, foul, with small and large hard curds.	.052	10.7	41.4	12.8	7.59
Mostly watery, granular, with fine white curds.	.049	13.3	52.6	8.8	7.48
Very watery, greenish, with large, hard curds, soft curds and mucus.	.054	13.6	50.5	12.6	10.15
Same as in No. 33.....	.059	13.8	45.6	9.0	10.72
Part formed, partly thin, pasty, fermentative, mucus.	.058	14.3	46.4	17.2	9.67
Watery, greenish, small soft curds.	(.05)	16.0	.....	8.0	2.80
Constipated, part formed and rather hard, part thin and smooth.	.038	17.6	44.1	9.2	1.30

the nitrogen of the amino acids, prolin, oxyprolin, trytophan, arginin, and histodin, which have all or part of their nitrogen in forms other than NH_2 groups.[4] Accordingly, if we multiply by 1.33 the amount of amino nitrogen found after acid hydrolysis of a mixture containing either animal proteins or their digestion products, we obtain approximately the amount of nitrogen present as protein and proteolytic products. Applying this factor (1.33) to our results for amino nitrogen after hydrolysis, we find that the feces on the average contain from 60 to 70 per cent. of their nitrogen in these forms. The

TABLE 2.—CASES FOLLOWED—

Case	Condition	Date	Food
E. V., 5½ mos.	Convalescent from diarrhea; doing well. Weight fluctuating, but rising on whole.	November 6..	2-4-1.8 with barley and malt soup extract
		November 7..	2-4-1.8 with barley and malt soup extract
		November 8..	2-4-1.8 with barley and malt soup extract
		November 9..	2-4-1.8 with barley and malt soup extract
		November 10..	2-4-1.8 with barley.....
		November 11..	2-4-1.8 with barley.....
		November 12..	2-4-1.8 with barley.....
E. W., 12 mos.	Convalescent from pneumonia. Gaining weight.	December 19..	3-5-2.8 with beef juice and cereal.....
		December 20..	3-5-2.8 with beef juice and cereal.....
		December 21..	3-5-2.8 with beef juice and cereal.....

TABLE 3.—BREAST—

Case	Condition	Date	Food
G. R., 4 mos.....	Normal	December 22.....	Breast fed .....
A. R., 5 mos....	Marasmus, gaining slightly.....	January 29.....	Breast fed .....
J. I., 3 mos.....	Marasmus, gaining slightly.....	Jan. 30-Feb. 2...	Breast fed .....
		February 2-5.....	Breast fed .....

free amino acid nitrogen alone constitutes from 2.4 to 24 per cent. of the total, while proteins, and possibly higher intermediate products, make up the balance.

The *urea* determinations were not performed on the same stools as the other determinations, for the reason that when the latter were done the urea method had not been entirely perfected. We have, however, determined the proportion of urea nitrogen in a separate lot of stools and present the results in table 4. They show that the proportion of the total nitrogen in the form of urea varies between 9 and 5.6 per cent. Nineteen out of the 25 stools contained no urea at all. That its absence

4. Van Slyke: Jour. Biol. Chem., 1911, x, 15; 1912, xii, 295.

was due to bacterial transformation of urea into ammonia appears unlikely, because some of the stools which contained no urea also contained but little ammonia. It should be stated that unless the separation between stools and urine is most carefully supervised, a high urea figure must be questioned.

The *ammonia* varies between 4 and 37 per cent. of the total fecal nitrogen. It usually equals or exceeds the amino acid nitrogen. This fact gives a definite basis for the accepted opinion that the fecal ammonia is of bacterial origin. When animal proteins, such as casein,

—ON SUCCESSIVE DAYS

Stools	Gm. Total N. Per Gm. Dried Wt.	Per Cent. of Total N. as			Acidity c.c. N/10 NaOH
		Free NH_2	NH_2 After Hydrolysis	Free NH_3	
Part formed, granular. Fine white, curds.	.053	8.4	49.0	4.7	3.74
Part formed, granular. Fine white, curds.	.058	6.8	52.5	4.3	3.97
Mostly formed	.054	3.3	49.7	3.6	2.57
Softer	.051	7.2	50.0	5.2	7.92
Softer, large soft curds	.051	6.6	47.7	7.6	7.95
Mostly formed again, though soft.	.073	4.8	43.4	6.9	3.13
Entirely formed; hard and soapy	.071	1.8	46.9	8.1	0
Watery; curds, acid, mucus	.052	10.7	41.4	12.8	7.59
Watery; curds, acid, mucus	.059	13.8	45.6	9.0	10.72
Watery; curds, acid, mucus	.054	13.6	50.5	12.6	10.15

—FED INFANTS

Stools	Gm. Total N. Per Gm. Dried Wt.	Per Cent. of Total N. as			Acidity c.c. N/10 NaOH
		Free NH_2	NH_2 After Hydrolysis	Free NH_3	
Watery, soft curds, yellow-green	.046	2.7	49.7	5.2	15.16
Partly formed	.046	6.2	50.6	7.8	14.59
Watery, fat curds	.065	6.3	47.6	5.9	9.65
Watery, fat curds	.059	6.4	47.4	3.8	10.61

are split by non-bacterial agents, such as acids, or aseptic digestion with trypsin, the ammonia nitrogen is less than one-seventh the free NH_2 nitrogen.

No relation is perceptible between the proportion of ammonia and the condition of the patients or the nature of the stools. The amount of free amino acid nitrogen also bears no evident relation to the ammonia or to the acidity of the stools. The figures for each vary independently.

Nor does the acidity of the stools bear any observable relation to the ammonia. From the theory that fermentive and putrefactive bacteria are antagonistic, one would expect low ammonia when the acidity is high, and vice versa. It will be seen, however, that while this rela-

TABLE 4.—UREA DETERMINATIONS

Name	Food	Description of Stool	Total N Per Gm. Dried Wt.	Ammonia N. Per Gm. Dried Wt.	Ammonia N. Per Cent. of Total N.	Urea N. Per Gm. Dried Wt.	Urea N. Per Cent. of Total N.
J. D., 6 mos.	1.6-4-1.4 with barley	Soft, pasty	.0390	.00054	1.4	0	0
V. C., 8 mos.	1.8-2.2-2 with barley	Watery	.0302	.00062	2.0	.00033	1.1
F. B., 5 mos.	Protein milk with rice flour and cane sugar.	Formed	.0375	.00130	3.6	0	0
		Part formed, part watery	.0396	.00060	1.4	0	0
F. S., 7 mos.	2-4-1.8 with barley	Mostly watery	.0488	.00055	1.1	0	0
A. A., 7 mos.	1.6-5-2 with rice flour	Watery and pasty	.0493	.00100	2.2	0	0
Ach. A., 7 mos.	Protein milk with barley	Mostly formed	.0371	.00080	2.2	0	0
		Part formed, part pasty	.0373	.00070	1.8	0	0
A. R., 8 mos.	Protein milk with barley and beef juice.	Formed	.0445	.00170	3.8	0	0
J. F., 9 mos.	2-5-1.8	Soft, pasty, green, mucus	.0682	.00429	6.3	.0011	1.6
J. F., 9 mos.	2.6-5-2.4	Pasty, greenish-brown, foul; mucus.	.0608	.00433	7.1	0	0
M. R., 4 mos.	1.8-6-2.4 with barley	Soft, pasty and watery, pale yellow.	.0632	.00458	7.3	0	0
N. M., 7 mos.	Protein milk with barley	Mostly soft pasty, part watery, may be urine.	.0573	.00609	10.6	.00322	5.6
N. M.	Protein milk, 5 feedings; 2.6-5-2.4 with barley, 2 feedings.	Formed, but soft; mucus and bean curds.	.0403	.00307	7.6	0	0
S. J., 9 mos.	Protein milk	Formed, pale yellow, part hard.	.0367	.00198	5.4	0	0
R. A'H., 5 mos.	Protein milk with barley and cane sugar, 5 feedings; breast milk, 2 feedings.	Watery, pale yellow, with curds and mucus.	.0453	.00487	10.7	.00117	2.6
P. K., 4 mos.	2.8-5-2.6	Formed but soft, pale; bean curds.	.0504	.00383	7.6	0	0
M. L., 17 mos.	Soft diet	Part formed, part watery; mucus	.0737	.00392	5.3	0	0
W. C., 3 mos.	Protein milk with barley and cane sugar.	Pasty, smooth; mucus	.....	.....	9.7	0	0
A. H., 2 mos.	1.6-5-2.0	Very watery with fat curds and mucus.	.1026	.00724	7.1	0	0
L. A., 4 mos.	Breast milk; one feeding of protein milk; with olive oil.	Very watery; oil floating on surface, bright yellow.	.0183	.00395	21.6	.00103	5.6
R. P., 5 mos.	2-5-1.8 with barley; breast milk, two feedings.	Part pasty, part watery; many fat curds.	.0513	.00236	4.6	0	0
C. S., 3 mos.	1.8-5-1.6, four feedings; breast milk, three feedings.	Pasty; portion formed and hard; mucus.	.0462	.00179	3.9	0	0
E. K., 6 mos.	1.2-4-2.2	Part formed, constipated; part pasty, dark brown, smooth.	.0386	.00351	9.1	.00074	1.9

tionship exists in some cases, in others both ammonia and acidity are low, and in still others both are fairly high. In a few cases, such as No. 25, where a high ammonia content is accompanied by a low acidity, calculation indicates that the lack of acidity is due to the neutralization of acid by the unusual amount of ammonia, rather than to lack of acid formation.

SUMMARY

Of the total nitrogen of infants' feces, from 50 to 70 per cent. was found as proteins and amino acids, 2.4 to 24 per cent. as free amino acids, and from 3 to 37 per cent. as ammonia. Usually, though not invariably, the higher percentages of amino acid nitrogen were found in the looser stools, quicker passage through the intestine resulting in somewhat less complete absorption of these protein digestion products.

The results show no evidence of antagonism between the processes of acid and ammonia formation respectively in the intestine.

Urea was certainly absent in three-fourths of the stools examined, and in the other fourth formed only from 1 to 5.6 per cent. of the total nitrogen. Despite the care taken to keep the feces free from urine, we do not feel certain that at least a part of the few positive results may not have been due to the admixture of traces of urine. As some even of the watery stools contained no urea, it appears improbable that the intestine is capable of rendering significant assistance to the kidney, at least in the excretion of urea.

PROGRESS IN PEDIATRICS

PROGRESS IN OTOLOGY

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Were progress in otology to be reviewed from the standpoint of new discoveries during the past year, there might be but little to record; and even if there had been numerous inventions, there still would be but little at this time to relate or to advise concerning their importance as progressive factors.

Medical progress in any line is not a matter of innovations so much as it is degree in the dissemination of methods and principles that through careful analysis and thorough investigation have proven worthy of adoption. In that sense, then, it is probable that greater advancement has been made in the subject of otology during the past year than in any like period before. The past decade has marked a particularly active period in the recognition by specialist and general practitioner alike of important facts pertaining to the care of ears, points in prophylaxis, early recognition of pathologic processes and safety precautions in the course of their treatment. But the burden of responsibility for the preservation of the sense of hearing rests more heavily on the family physician or the pediatrist, whose position is that of chief adviser through the years of greatest susceptibility to ear infections. Probably nine-tenths of all cases of acquired deafness that come under the specialist's care in their adult years originated in childhood from preventable causes.

Middle ear suppurations, which have so frequently complicated the acute infectious diseases of children, we have learned, are indeed rare if the child's nares are kept freely open and cleansed by the careful employment of mild astringent applications to the turgescent mucous membrane, and the nasopharynx is kept clear of all accumulating mucous discharges. All cleansing technic in the course of acute processes should include only such agents as are capable of being applied with pledgets of cotton rather than forced sprays or irrigations of watery solutions in the nose and nasopharynx, which have doubtless caused more ear infections than they have ever prevented. The nasal douche, the safest of all irrigating methods, should be employed only after the swollen intranasal membrane has been reduced by astringent applications, and even then with greatest care if there be a purulent discharge from any accessory sinus.

One of the most important factors working to-day toward the insurance of good ears for the next generation, or all future generations, is the widespread or almost universal recognition of the effects of toxic absorption from necrotic tonsils and the consequent necessity of tonsillar enucleation as early in childhood as the diseased conditions are observed. Surely some one, and at some not far distant day, we trust, will discover a reason for the almost universal degenerative process which invades the faucial tonsils, most frequently in childhood, yet from which no age is immune, causing the secretion in the crypts to coagulate and become the highly toxic necrotic concretions that, reabsorbed, pass into the lymphatics to poison not only the contiguous tissues, but to affect vital functions throughout the body. Whatever might have been the gland's original function, it is lost or at best suspended in the presence of such a pathologic condition, and the dangers of retention are too great to warrant too prolonged attempts at restoration; so there is left but the one satisfactory alternative, enucleation. Even chronic aural discharges are frequently found to cease within a few days after the enucleation of tonsils that were perhaps so small that their necrotic state was for a long time unsuspected; in fact, it is the hidden, submerged, *retention* tonsil, regardless of its size, that is capable of the greatest damage to the ears. Remnants of tonsils, left in the fossae by incomplete operations, are also capable of prolonging all the ill effects that the original whole tonsil had created, which fact might explain myriads of disappointments where tonsils were supposed to have been removed. Pathologic enlargement of adenoid tissue in the nasopharynx, always an important factor in the interference of aural function, is probably due in most cases to toxic absorption from necrotic tonsils; hence the recurrence of adenoids in cases in which the removal of tonsils was either not attempted, or else attempted and not perfectly accomplished. Acute suppurative ethmoiditis is one of the most frequent sources of otitis media and should be guarded against in all severe or supposed prolonged "colds in the head." Every purulent nasal discharge, with but few exceptions, is from one or more of the accessory sinuses and demands immediate attention.

In the treatment of suppurative sinus and otitic infections one of the most important advances in recent years is vaccine therapy, to which the past year's testimony adds strong support. Difficulties attending the procuring of perfect autogenous vaccines have brought about the stock products which are now in most general use. Arguments against the use of the mixed vaccines have been refuted by clinical results, for when used in conjunction with and not as a substitute for the usual local and constitutional measures as employed by the up-to-

date aurist and rhinologist, the course of disease in an average case seems to be shortened by at least one-half, while even in those in which no benefit is observed, no harmful effects can be detected. A measure, then, that can be employed with probable benefit in 75 per cent. of all suppurative processes in the nasal accessory cavities or in those of the temporal bone, with a reasonable assurance that at least no harm will result from its use in the other 25 per cent., is indeed a safe and valuable remedy and is to be regarded as one of the most important advances of the age. Conservatism in the treatment of mastoiditis has been gaining ground for the past ten years, and particularly so with the more general adoption of vaccine therapy. The day may not be far distant when the radical mastoid operation will be a rare occurrence even in chronic suppurations, and entirely unnecessary in acute or subacute involvements. This change is being brought about by a more widespread knowledge concerning the treatment of conditions that lead to mastoid disease and by the improved means at our command for the treatment of the fully developed complication. Vaccine therapy is to-day one of the most potent instruments in the hand of preventive medicine, not so much in the sense of an immunizer against the primary invasion of disease-producing bacteria, but rather as a means of cutting off their advance into other surrounding and probably more vital tissues. In no part of the body is this more true or more evident than in the nose and throat where an unchecked apparently simple infection may lead to a serious impairment of one or all of the special senses, smell, taste, hearing and sight.

With the prevention or cure, then, of local infections that might eventually lead to middle ear suppuration, there is also prevented a large percentage of intracranial abscesses and meningeal complications.

During the past year there has continued some most creditable original research in disease of the labyrinth and methods of treatment. Such involvements, however, are more difficult of detection, require more intricate technic in treatment than the majority of diseases affecting only the middle and outer ear, and come only within the province of the skilled specialist, though every practitioner should remember that suppurative disease of the labyrinth is always secondary to an acute or chronic otitis media, and is apt to be accompanied by sudden, total and permanent deafness.

Indiscriminate efforts to inflate the middle ear, however attempted, or for whatever purpose practiced, is a process capable of marked and lasting injury to the ears and can not be too frequently nor too severely criticized. The forcing of air or other vapors into or through the eustachian tube, is a safe procedure only when the physician has thoroughly prepared the nares, nasopharynx and pharynx for its introduction, and even then its indications are exceedingly rare.

The employment of bougies for reestablishing a clear opening through the eustachian tube and to preserve its integrity has in a large measure been disappointing.

But in the treatment of chronic, nonsuppurative catarrhal lesions of the ears the greatest patience is required at all times, for even under most favorable circumstances results come slowly, and to know just how long a certain line of treatment should be persisted in or when discarded, is still at times a perplexing problem, and some things must be recognized as impossible of accomplishment.

A popular impression with the laity and not infrequently among physicians that little or nothing can be done in the way of treatment for deafness has been established from erroneous opinions or absolute ignorance regarding the varieties of deafness and the various causes leading thereto. Such an opinion is harmful, in that it leads to neglect through the stages when relief might readily be obtained. The majority of cases of defective hearing are of a slowly progressive type, due to causes easily removable or perfectly amenable to cure in the earlier stages of auditory disturbance.

The organ of hearing is indeed a wonderfully delicate mechanism, and capable of easy and irreparable injury, for which very reason nature seems to have placed it in the most perfectly fortified position. The eustachian tube and the external auditory canal are practically the only two roads open to attacks by infection and the latter of these but slightly exposed. If, then, there can be brought about a more concerted effort on the part of specialists and family physicians to educate the laity to the fact that nearly all diseases of the ear are secondary to infections in the nose and throat, that the earliest symptom of an ear involvement should serve as an alarm to seek immediate relief, and that "deafness can best be cured by being prevented," future generations will hear and regard as selfevident truths, principles and precautions which we have been slow to understand and still less willing to adopt.

The following list is not intended to be a complete index of the past year's literature on otology, but as representative selections bearing some relation to the subject in its various phases. From these herein given, however, one can not help but be impressed with the deep interest thus manifested, which in itself is one of the most potent factors for progress.

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